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PREVENTION OF TRAUMATIC DEAFNESS

A PRELIMINARY REPORT

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Loss of hearing due to acoustic trauma has been established as a clinical entity. It has also been established that no known form of therapy can improve this type of hearing defect. After rest has removed what fatigue factor existed, the remaining damage may be considered permanent. With this in mind, it is clear that energy must be directed toward the prevention of deafness in persons not yet affected and the arrest of progress in those who have already suffered some loss.

The simplest means of decreasing the intensity of sound reaching the ear has always been the plugging of the external auditory canal. Chippendale,[1] in 1866, suggested the use of cotton. Cousins [2] recommended the use of ear plugs made of india rubber. During the first World War it was found that cotton soaked in glycerin afforded the best protection.[3] This soaking of the cotton in some viscous substance is still held by many others [4] to be essential for maximal dampening. Vadala [5] described an ear protector made of porous rubber, the body of which fits in the concha of the ear, with a small projection extending into the cartilaginous canal. He stated that there is a dissipation of the sound waves due to the multiplicity of the holes in the porous rubber.

The use of a pad over the entire external auricle has been recommended by several writers.[4b] This device is particularly feasible for aviators, as the pads may be sewn inside the helmet.

Dickson and associates,[6] in a thorough study of protective devices for the ears, found that the most effective single means was an ear plug composed of some solid, but pliable, substance. A soft plastic capable of readily taking the size and shape of the individual meatus was used. Such an ear plug afforded the wearer 25 decibels of protection. An aviator's helmet fitted with either telephone ear pieces or pads was found to afford 20 decibels of protection.

The value of these various means of closing the ears is questionable in spite of their many recommendations. Dickson and co-workers admitted that ear pro-

1. Chippendale, J.: Deafness Produced by Reports of Firearms, Correspondence, Lancet **2**:170 (Aug. 11) 1866.

2. Cousins, W., in discussion on Barr, T.: Injurious Effects of Loud Noises on the Hearing, Brit. M. J. **2**:675 (Sept. 20) 1890.

3. Richardson, C. W.: Ear Protectors, Tr. Am. Otol. Soc. **14**:525-542, 1918.

4. (*a*) Blake, C. J., in Kober, G. M., and Hayhurst, E. R.: Industrial Health, Philadelphia, P. Blakiston's Son & Co., 1924, pp. 898-904. (*b*) Ranken, D.: Occupational Diseases of Ear, Nose and Throat, and Their Prevention, Brit. M. J. **2**:891-892 (Nov. 14) 1925. (*c*) McCord, C. P.; Teal, E. E., and Witheridge, W. N.: Noise and Its Effect on Human Beings, J. A. M. A. **110**:1553-1560 (May 7) 1938.

5. Vadala, A. J.: Effects of Gun Explosions on the Ear and Hearing Mechanism, Mil. Surgeon **66**:810-822 (June) 1930.

6. Dickson, E. D. D., and Ewing, A. W. G.: The Protection of Hearing, J. Laryng. & Otol. **56**:225-242 (July) 1941.

tectors do not decrease the intensity of low tones to the extent that they do the intensity of high tones, with the result that the former may still cause damage. One must also consider the possible role played by bone-conducted sound and vibration. Ear protectors have always been unpopular with laborers, in that they are bound to entail both discomfort and inconvenience. Employers also recognize the increased accident hazard, in that men with plugged ears may well not hear the warning of impending danger from their fellow-workers.

Since it has been shown that the lowering of air-conducted intensities does not eliminate the danger of traumatic deafness, some investigators have turned to the dampening of vibration before it reaches the body as a possible aid. Spring-cushioned platforms and benches have been recommended and put into use.[7] The value of rubber mats and soles is generally recognized.[8]

The most logical approach to the entire problem is the elimination of noise at its source. In late years definite strides have been taken in this direction. Welding may replace riveting. The revolving parts of machinery may be kept in balance and worn parts replaced. Much may be accomplished by reducing resonance wherever possible. This must start with the architectural design. Sound-absorbent materials on walls, ceilings and floors will reduce noise 5 to 10 decibels.[4c] While much has been done, and much may still be accomplished along this line, it must be admitted that the noise of industry has increased even more rapidly than it has been eliminated.

Temkin [7b] expressed the belief that the length of exposure to noise in one day is important and recommended shorter hours. He suggested the rotation of men between noisy and noiseless occupations within the same factory. Gilbert [8b] also held that this plan was ideal but stressed its impracticability because of the necessity of taking skilled labor away from specialized lines of work. He suggested the changing of men to different machines or positions in the same room, however, in that the difference in intensity, resonance and pitch encountered would be of some benefit.

Since some men are more predisposed to traumatic deafness than others, the elimination of such persons from noisy industry would seem to be an effective preventive. Certain to suffer are persons with a hereditary history of deafness. There is a controversy over the susceptibility of persons possessing either a scarred membrana tympani and ossicles or active chronic infection of the middle ear. Experimental evidence supports each side. Temkin [7b] expressed the belief that persons with conduction deafness should not work at noisy occupations in which vibration also predominates. The fact that stapes fixation does actually increase the intensity of bone-conducted stimuli upholds this contention.

Peyser,[9] stressing the need of prophylaxis from the standpoint of occupational deafness in industry, recently devised an audiometric test whereby the resistance of a person's ears to loud sounds may be determined. Two threshold values for an ear for a frequency of 256 cycles are compared. The first is taken before, and the second thirty seconds after, an 80 decibel tone at that frequency is applied

7. (*a*) Swann, C. C.: The Effect of Noise on Hearing, Internat. J. Med. & Surg. **46**: 314-315 (July) 1933. (*b*) Temkin, J.: Injury to Ears Due to Noise and Vibration, Monatschr. f. Ohrenh. **67**:257 (March); 450 (April); 527 (May); 705 (June); 823 (July) 1933. (*c*) McCord and others.[4c]

8. (*a*) Scal, J. C.: Noise Deafness in Industry and Environment, New York State J. Med. **37**:1251-1254 (Nov. 1) 1933. (*b*) Gilbert, D. J.: Influence of Industrial Noises, J. Indust. Hyg. **3**:264-275 (Jan.) 1922. (*c*) McCord and others.[4c] (*d*) Swann.[7a]

9. Peyser, A.: Zur Methodik einer otologischen Prophylaxis der industriellen Lärmschwerhörigkeit, Acta oto-laryng. **28**:443-462, 1940.

for five minutes. Of 80 subjects so examined, 7 showed a rise in threshold of over 5 decibels after stimulation. These persons, he concluded, had a definite lack of resistance to loud sounds and would acquire serious impairment of hearing if exposed over any period. Unfortunately, none of Peyser's subjects were later exposed to any acoustic trauma; so his contention remained just that. The value of such a test is quite apparent, and the theory behind it is sound.

Many studies have been made on auditory fatigue as such. Hughson and Witting,[10] working with animals, found that any frequency throughout the entire normally tested range, if of sufficient intensity, will cause fatigue. The maximum period required for complete recovery in their series was seventy-two hours.

With human subjects, Smith [11] found that after exposure to a tone of 2000 cycles at an intensity of 100 decibels for two minutes the greatest fatigue occurred at a frequency of 4000 cycles. The fatigue variance was all the way from 10 to 56 decibels at that frequency, while at the exposure frequency the loss, though definite, was less. At 1000 cycles it was negligible. He advanced the theory that it is the lengthening of the refractory period of the individual nerve fibers by the extreme intensity that causes fatigue. This increased refractory period results in a relative decrease in the number of working fibers at any particular moment, with a consequent decrease in the sensitivity itself. In a later work he [12] concluded that there must also be involvement of the higher centers.

That the irreversible lesions of traumatic deafness are atrophy of the auditory nerve and degeneration of the organ of Corti was given considerable support by the studies of Crowe, Guild and Polvogt.[13]

Kobrak, Lindsay and Perlman [14] subjected a person with a normal hearing curve to a tone of 1024 cycles at a 95 decibel intensity for two minutes. Immediately at the end of this period there was a rise in threshold of 15 decibels at a frequency of 1024 cycles, which the authors reported as the only frequency for which any change was shown. Twenty seconds later the threshold was 12.5 decibels above normal. At the end of sixty seconds it was 5 decibels, and at the end of two and a half minutes it had returned to normal.

These authors contended that acoustic fatigue is a protective mechanism, decreasing the sensitivity of the ear during loud sounds—an acoustic adaptation that may be likened to the light-dark adaptation of the eye. Such an adaptation would be for prolonged stimuli, as compared with the sudden stimuli which the muscle reflexes of the middle ear guard against.

Perlman [15] exposed young adults to a fatiguing tone just short of the threshold of pain for one hour. While each subject was exposed to but one frequency, all octaves from 64 to 8192 cycles, with the exception of 128 cycles, were tested for. When a rise in threshold was seen, it generally reached a maximum one octave

10. Hughson, W., and Witting, E. G.: An Objective Study of Auditory Fatigue, Acta oto-laryng. **21**:457-486, 1935.

11. Smith, A. F. R.: Auditory Fatigue, Brit. J. Psychol. **25**:77-85 (July) 1934.

12. Smith, A. F. R.: Experimental Deafness, Brit. J. Psychol **26**:233-244 (Jan.) 1936.

13. Crowe, S. J.; Guild, S. R., and Polvogt, L. M.: Observations on the Pathology of High-Tone Deafness, Bull. Johns Hopkins Hosp. **54**:314-379 (May) 1935.

14. Kobrak, H. G.; Lindsay, J. R., and Perlman, H. B.: Experimental Observations on the Question of Auditory Fatigue, Laryngoscope **51**:798-810 (Aug.) 1941.

15. Perlman, H. B.: Acoustic Trauma in Man, Arch. Otolaryng. **34**:429-452 (Sept.) 1941.

above the fatiguing tone. This held for all fatiguing frequencies between 512 and 4096 cycles inclusive. No fatigue was caused by a tone of 64 cycles. The results for 256 cycles were not uniform, and, because of technical complications, the determinations for 8192 cycles were not considered accurate.

EXPERIMENTAL STUDY

The vast number of men being inducted into the Army at present affords a fertile field for study of the relation of auditory fatigue to irreversible hearing loss caused by acoustic trauma. The present study was carried out at a quartermaster replacement training center, where all enlisted personnel are new inductees, having never fired army weapons of any description.

Method.—The men tested were principally selected at random from various companies. Some were patients who presented themselves at the eye, ear, nose and throat clinic at the station hospital. For the most part these subjects had ophthalmic disturbances, as men with complaints referable to the ears, nose and throat that might in any way affect the results of the study were not tested.

With Peyser's work as a basis for further study, his fatigue tone of 256 cycles at an 80 decibel intensity was used. Of the 85 subjects in the series, 52 were exposed to this fatigue tone for five minutes in each ear and the remaining 33 to the same tone for eight minutes. All fatiguing tests, as well as all audiograms, were made with a Western Electric no. 6a audiometer. Readings were recorded at 5 decibel intervals. The closest multiple of 5 to the reading obtained was recorded. While a soundproof room was not available, a relatively quiet room in a remote part of the hospital was set aside for the testing.

A short history was taken first for each subject. The subject was questioned with regard to deafness in the family, whether he had ever fired a gun or worked in a noisy place, whether he had at any time noted tinnitus and whether he had ever had otitis media. The ears of each subject were examined by means of an ear speculum and a head mirror.

An audiogram was then obtained for each ear. After this, the right ear was exposed to the 256 cycle fatiguing tone at an intensity of 80 decibels for either the five or the eight minute period. One minute was then allowed to elapse, after which a complete audiogram was taken for the exposed ear. The left ear was then fatigued and tested in the same manner.

While Peyser determined his prefatigue and postfatigue thresholds only for the frequency of his fatiguing tone, that is, 256 cycles, it was felt that much more information could be gathered if complete audiograms were made both before and after exposure to the fatiguing tone.

All the men tested were instructed to report back the morning after they completed their basic firing on both the pistol and the rifle range. In no instance were they known to have gone out on the range sooner than one week after being exposed to the fatiguing tone.

Their course of firing consisted of 12 rounds with a .45 caliber pistol and 70 rounds with the .30 caliber rifle, of which 40 rounds were rapid fire and 30 rounds slow fire. In addition to the firing of their own weapons, they were subjected to the acoustic trauma of the firing of other arms in close proximity. Twenty-eight men of the series also coached. A firing coach stands to the right and just in front of his pupil. In such a position he is more exposed to the muzzle blast of the gun than if he were actually firing. Time spent on the firing range varied from two to three days.

Between the firing and the next audiometric study there was a minimum lapse of fourteen hours and a maximum lapse of twenty-two hours. Exceptions to this were the cases of 3 men who returned thirty-six hours, 1 three days and 1 five days later.

On their return the subjects were asked whether they used ear plugs of any sort, whether they coached as well as fired and whether they noted deafness, tinnitus or vertigo after firing.

Complete audiograms were again made on all subjects as they returned. Those who showed an increase over the originally determined threshold of 10 decibels or more for any one or more tested frequencies were instructed to return in seven days. Those who still showed a loss at the end of this period were asked to return again in another week.

Observations.—The average age of the 85 subjects was 27.2 years. Nine soldiers gave a history of deafness in the family. In each instance, however, but

TABLE 1.—*Data on the Series of Eighty-Five Subjects*

Subject No.*	Family History of Deafness †	History of Otitis Media ‡	Scarred Membrana Tympani §	Coached	Cotton in Ears While Firing ‖	Subjective Deafness After Firing #	Tinnitus After Firing #	Fatigue Period, Min.
1	—	—	++	—	—	++	++	5
2	—	—	—	+	½+	++	++	5
3	—	—	—	+	—	+	—	5
4	—	—	—	—	—	—	++	5
5	—	—	—	+	—	—	+	5
6	—	—	++	+	—	—	+	5
7	—	—	—	—	—	+	++	5
8	—	R	+	—	—	—	—	5
9	—	—	—	—	—	—	+	5
10	—	—	+R	—	—	—	—	5
11	—	—	—	—	—	—	—	5
12	M	R & L	+++	+	—	—	+	5
13	—	—	—	+	—	—	—	5
14	—	—	—	+	—	—	—	5
15	—	—	++	+	—	+	+	5
16	—	L	++	—	—	—	—	8
17	M	—	+	—	—	—	—	8
18	—	—	—	—	—	—	—	8
19	—	—	—	—	+	—	—	8
20	—	—	—	—	+	—	++	8
21	—	—	—	—	—	—	++	8
22	—	R	—	—	+	—	+	5
23	F	—	—	—	—	—	—	5
24	F	—	++	—	—	—	—	8
25	—	—	—	—	—	—	—	8
26	—	R	+++	—	—	—	—	8
27	—	—	—	—	—	—	—	8
28	—	—	—	+	½+	+	+	5
29	—	—	+	—	—	+	+	5
30	—	—	—	+	—	—	—	5
31	—	—	—	—	—	+	+	5
32	—	—	++	+	—	—	+	5
33	—	—	—	—	—	—	++	5
34	—	—	++	+	—	+	++	5
35	—	—	—	+	—	+	+	5
36	—	—	+	—	—	—	+	8
37	—	—	—	—	—	—	—	8
38	—	—	++	+	—	—	—	5
39	—	—	—	—	—	—	+	5
40	—	—	—	+	—	—	—	5
41	—	—	—	—	—	—	+	5
42	—	—	—	+	—	—	—	5
43	—	—	—	+	—	—	—	5
44	—	—	—	+	—	—	+	5
45	—	—	—	+	—	—	—	5
46	—	—	—	+	—	—	—	5
47	F	—	—	—	+	—	—	5
48	—	—	—	—	—	—	+	5
49	—	—	—	—	+	—	—	5
50	—	—	++	+	—	+	+	5
51	—	—	—	+	½+	—	—	5
52	—	—	+R	+	—	—	—	5
53	—	—	—	+	—	—	+	5
54	—	—	—	+	—	—	+	5
55	—	—	+	—	—	—	—	5
56	M	—	—	—	—	—	—	5
57	—	—	—	—	—	—	—	5
58	S	—	—	—	—	—	—	5
59	—	—	—	—	—	—	—	5
60	—	—	—	—	—	—	—	5
61	—	—	—	—	—	—	—	5
62	—	—	—	—	—	—	—	5
63	—	—	+++	—	—	—	—	5
64	—	—	—	—	—	—	—	5
65	—	—	—	—	—	—	++	8
66	—	R	++R	—	—	+	++	8
67	—	—	—	—	½+	—	—	8
68	M	—	—	+	—	—	+	8
69	—	—	—	—	+	—	—	8
70	—	—	—	—	½+	—	—	8
71	—	—	+	—	—	—	+	8
72	—	—	—	+	—	—	—	8
73	F	—	—	—	½+	—	—	8
74	—	—	—	+	—	+	+	8
75	—	—	++	—	—	—	—	8
76	—	R	++	—	—	—	—	8
77	—	—	—	—	—	—	—	8
78	—	—	++	—	—	—	—	8
79	—	—	—	—	—	—	—	8
80	—	—	—	—	—	—	—	8
81	—	—	++R	—	—	—	—	8
82	—	—	—	—	—	—	—	8
83	—	—	—	—	—	—	—	8
84	—	—	—	—	—	—	—	8
85	—	—	—	—	—	—	—	8
Total	9	7	25	28	12	13	32	

* Cases have been renumbered to facilitate grouping.

† M indicates mother; F, father, and S, sister.

‡ R indicates the right ear, and L, the left ear.

§ The number of +'s indicates the degree of scarring on the basis of + to ++++ When it is not otherwise designated, scarring is bilateral.

‖ ½+ means part time.

+ means subjective complaint of deafness or tinnitus after firing; ++, subjective complaint still present next morning.

TABLE 2.—*Rise Above Original Threshold Curve, Expressed in Decibels (a) After Fatiguing, (b) After Firing and (c) After Recovery for One to Two Weeks**

Subject No.		Frequencies																	
		128		256		512		1024		2048		4096		5792		8192		9747	
		R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L
		Threshold Curves Correctly Predicted to Rise																	
1	(a)	..	..	10	10	10	15	10	15	..	15	..	..	..	10	..	..	..	..
	(b)	..	..	20	30	35	50	40	70	65	85	30	50	10	45	20	40	25	30
	(c)†	..	..	..	..	..	..	10	..	25	..	..	..	..	..	..	..	..	..
2	(a)	..	..	..	..	..	..	..	..	..	..	..	25	..	..	..	..	..	..
	(b)	..	..	..	..	..	..	..	..	..	..	15	40	..	..	..	..	..	..
	(c)†	..	..	..	..	..	..	..	..	..	..	10	40	..	..	..	..	..	..
3	(a)	..	..	..	..	15	..	10	..	..	..	..	..	..	..	..	..	10	..
	(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	10	..	..	..
	(c)	Did not return																	
4	(a)	10	..	15	..	..	..	10	..	..	..	..	10	..	..	..	..	..	..
	(b)	..	..	10	..	..	..	..	..	..	..	15	..	..	..	..	20	10	..
	(c)	Did not return																	
5	(a)	..	..	..	..	10	10	..	..	..	..	..	..	..	..	..	..	20	10
	(b)	15	10	15	10	20	15	..	..	..	..	..	..	10	..	..	10	30	10
	(c)	..	..	..	10	15	20	..	..	..	..	..	..	..	..	..	..	..	10
6	(a)	..	..	..	10	10	..	..	10	..	10	10	15	10	15	10	20	25	25
	(b)	..	..	..	..	..	..	..	..	..	..	15	..	..	..	10	15	15	25
	(c)	Did not return																	
7	(a)	..	..	10	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	(b)	..	..	..	..	..	..	..	..	..	20	20	15	15	25	20	25	10	40
	(c)	..	..	15	..	..	..	..	..	..	10	10	20	15	..	20	20	10	15
8	(a)	..	..	..	..	..	..	..	10	10	..	..	10	..	..	..	..	..	..
	(b)	25	20	25	20	35	30	55	45	65	35	50	40	50	35	45	45	55	55
	(c)	Did not return																	
9	(a)	..	..	..	..	..	..	..	..	..	..	..	..	10	..	..	..	..	..
	(b)	..	10	..	10	..	..	..	..	..	..	20	10	40	10	20	..	55	..
	(c)	..	10	..	..	..	..	..	..	..	..	20	10	25	10	10	10	55	..
10	(a)	10	..	10	..	10	..	..	..	..	..	..	..	..	..	..	..	..	..
	(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	10	..
	(c)	Did not return																	
11	(a)	..	..	..	..	..	..	..	..	..	10	..	10	..	..	..	..	..	..
	(b)	15	..	..	10	10	..	..	..	..	..	..	..	..	..	..	..	..	15
	(c)	Did not return																	
12	(a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	10
	(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	10
	(c)	Did not return																	
13	(a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	10	..	..
	(b)	..	..	..	..	..	..	..	..	..	..	10	..	..	..	..	10	..	..
	(c)	Did not return																	
14	(a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	10	..	..	..
	(b)	10	..	10	..	10	..	..	..	..	..	..	..	..	..	..	..	..	..
	(c)	Did not return																	
15	(a)	15	20	..	15	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	(b)	..	..	15	20	15	10	10	..	10	30	10	15	10	25	15	10	10	..
	(c)	Did not return																	
16	(a)	..	..	..	..	..	..	..	..	..	..	..	..	10	..	..	..	..	..
	(b)	..	..	..	..	..	..	10	..	..	..	..	..	15	..	..	..	..	..
	(c)	..	..	..	..	..	..	..	..	..	..	..	..	15	..	..	..	..	..
17	(a)	15	15	15	15	15	10	..	10	..	10	15	..	30	10	15	..	..	..
	(b)	15	..	10	10	..	10	10	15	15	10	10	10	25	..	20	..	..	..
	(c)	Did not return																	
18	(a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	25	..	30	..
	(b)	..	..	..	..	..	..	..	..	..	..	15	..	..	..	10	15	..	20
	(c)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	10
19	(a)	20	..	15	15	10	..	..	..	..	..	..	..	..	..	..	..	..	..
	(b)	..	..	..	..	..	..	..	..	..	10	..	..	..	10	..	..	..	..
	(c)	..	..	..	10	..	..	..	..	..	..	..	..	..	10	..	..	..	..
20	(a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	50	..	50
	(b)	..	..	..	..	..	15	..	..	..	..	..	25	..	45	..	45	..	30
	(c)	..	..	..	..	..	..	..	..	..	10	..	15	..	55	..	45	..	45
21	(a)	..	..	..	10	..	10	..	..	..	..	..	..	..	..	..	..	..	..
	(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	50	30	..	15	30
	(c)	..	..	..	..	..	..	..	..	..	..	..	..	..	35	20	..	..	..

* R indicates the right ear; L, the left ear.
† Subject tested after two weeks' recovery.

TABLE 2.—*Rise Above Original Threshold Curve, Expressed in Decibels (a) After Fatiguing, (b) After Firing and (c) After Recovery for One to Two Weeks *—Continued*

Subject No.		Frequencies																	
		128		256		512		1024		2048		4096		5792		8192		9747	
		R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L
Threshold Curves Predicted to Rise That Remained Stable																			
22	(a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	10	..	10	..
	(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	(c)	Not asked to return																	
23	(a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	20	..	..
	(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	(c)	Not asked to return																	
24	(a)	..	..	..	..	..	..	..	..	..	..	10	..	..	..	..	..	..	..
	(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	(c)	Not asked to return																	
25	(a)	..	..	20	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	(c)	Not asked to return																	
26	(a)	15	..	20	..	15	..	..	..	..	..	..	..	..	..	..	..	..	..
	(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	(c)	Not asked to return																	
27	(a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	10	..	..	..
	(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	(c)	Not asked to return																	
Threshold Curves Predicted Stable That Rose																			
28	(a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	55	..	10	25	15
	(c)	..	..	..	..	..	..	..	..	..	..	..	..	..	25	..	..	..	..
29	(a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	(b)	..	..	..	..	..	..	..	..	..	..	..	45	..	10	..	20	..	..
	(c)	Did not return																	
30	(a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	25	..
	(c)	..	..	..	..	..	..	..	..	..	..	..	10	..	..	..	..	..	..
31	(a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	(b)	..	..	..	..	..	..	..	..	..	..	..	10	..	..	..	..	..	..
	(c)	Did not return																	
32	(a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	(b)	..	..	..	..	..	10	..	..	..	..	20	..	..	..	..	..	..	..
	(c)	Did not return																	
33	(a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	(b)	..	..	..	..	..	..	..	..	..	..	25	..	..	..	..	..	..	..
	(c)†	..	..	..	..	..	..	..	..	..	..	20	..	..	..	..	..	..	..
34	(a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	(b)	..	..	..	..	..	..	..	..	..	..	40	10	70	40	50	..	25	15
	(c)†	..	..	..	..	..	..	..	..	..	..	..	..	15	..	..	..	..	..
35	(a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	(b)	..	..	..	..	..	..	..	..	10	..	10	..	..	10	..	..	..	..
	(c)	Did not return																	
36	(a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	30
	(c)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	10
37	(a)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	(b)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	15
	(c)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Threshold Curves Correctly Predicted Stable																			
38 to 85		No change in original threshold curve after exposure to fatiguing tone or after firing																	

one member was affected. There was no appreciable difference in the responses of these 9 men to the fatiguing tests and the trauma of firing and the responses of the whole series.

None of the men had at any time fired an army rifle. One of the series had previously worked in a canning factory. One had been a railroad fireman. One had worked in a steel mill. Two had run a tractor at some time. Thirty-one of the men had previously hunted with a shotgun or fired a .22 caliber rifle. Of these, 3 had also run a tractor, 2 had worked in a steel mill and 1 in a boiler factory.

Four men gave a history of previous attacks of tinnitus. All these subjects showed evidence in their audiograms of loss for high tones. Two also had some conduction deafness. The only significant fact in any of their histories was that 1 man with nerve deafness had previously done considerable hunting. Three of the 4 men showed definite fatigue, and these same 3 subjects had a rise in threshold when they returned from the firing range.

Seven men gave a history of having had one or more attacks of acute otitis media. Twenty-five subjects presented some degree of scarring of one or both membrana tympani. As a group, however, these men gave about the same responses in the tests as did the others.

It was necessary to establish the minimal loss in decibels which would be called a deviation from the normal, the normal for each subject being his original threshold curve. A deviation of less than 10 decibels was considered to be within experimental error. Any repeat audiogram showing a rise in threshold of 10 decibels or more above the original level for one or more of the tested frequencies was classified as indicating a loss. Of the total series, 27 soldiers fell in this category after being subjected to the fatigue tone. The remaining 58 were classified as not showing fatigue. This group of 27 men were expected to have a definite hearing loss on their return from the firing range at a later date. Of these, 21 did show such a loss according to the standard. Six of the group gave the same threshold curve after firing that they had given before. Of these, 1 had fired with cotton plugs in his ears.

Of the 58 men who had not shown fatigue, 10 had a rise in threshold on returning from the firing range. Forty-eight demonstrated no change. Eight of these 10 men had been exposed to the fatiguing tone for five minutes, while only 2 had been exposed for eight minutes. This is the only instance in the series of an appreciable difference in results for the two fatiguing periods.

The men who showed a hearing loss after firing were instructed to return seven days later. Of the 31 subjects who were instructed to return, only 16 did so, since many of the men were sent to other posts shortly after the completion of their basic course in firing.

Fifteen of these 16 subjects still showed some loss at the end of one or, if they returned still later, two weeks. For 5, a minimal rise in threshold was seen at frequencies at which previously it had not been demonstrated. In 1 case it was the only apparent remaining loss.

Of the 31 subjects showing after-firing loss, 10 complained of deafness after firing. Two of these still complained of deafness when audiograms were taken the next morning. Of the 54 subjects not showing after-firing loss, 3 complained of deafness after firing. None complained the next morning.

Of the 31 subjects showing after-firing loss, 19 complained of tinnitus after firing. Eight of these still complained of tinnitus the next morning. Of the 54 subjects not showing after-firing loss, 13 complained of tinnitus after firing. Two still complained of tinnitus the next morning.

One subject complained of vertigo after firing. It had disappeared by the next morning, but a considerable increase in his hearing threshold was still demonstrable.

Twelve of the men wore cotton plugs in their ears while firing. Six of these 12 men wore them only part of the time, however. For this group the percentage of rises in threshold after firing was approximately the same as that for the entire series.

That the average original threshold curve for the subjects who showed a rise after firing was higher than the average curve for those who did not may be seen in chart 1. This substantiates the argument that persons with impaired hearing are more predisposed to acoustic trauma.

Of the 27 subjects who showed loss of hearing after exposure to the fatigue tone, the number for whom fatigue was found at each tested frequency is demonstrated in chart 2. The same plan was followed for the 31 subjects who showed a rise in threshold after firing and for the 16 subjects who returned later for checks on the maximal recovery.

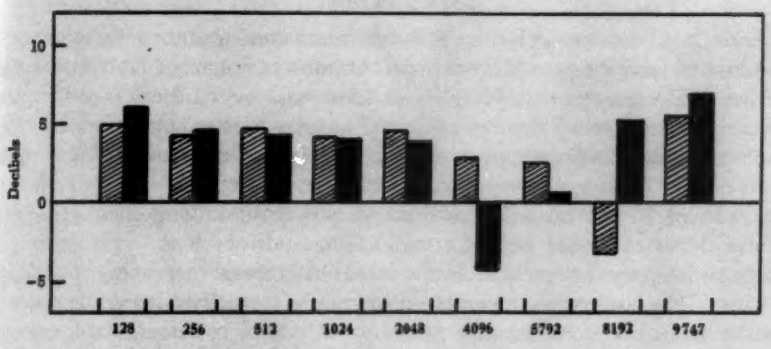

Chart 1.—Average difference in original thresholds of hearing between subjects later showing loss and those later showing no loss of hearing after firing, zero being taken as the threshold for the latter group. In this chart, and in chart 2, solid black indicates values for the left ear; cross hatching, values for the right ear.

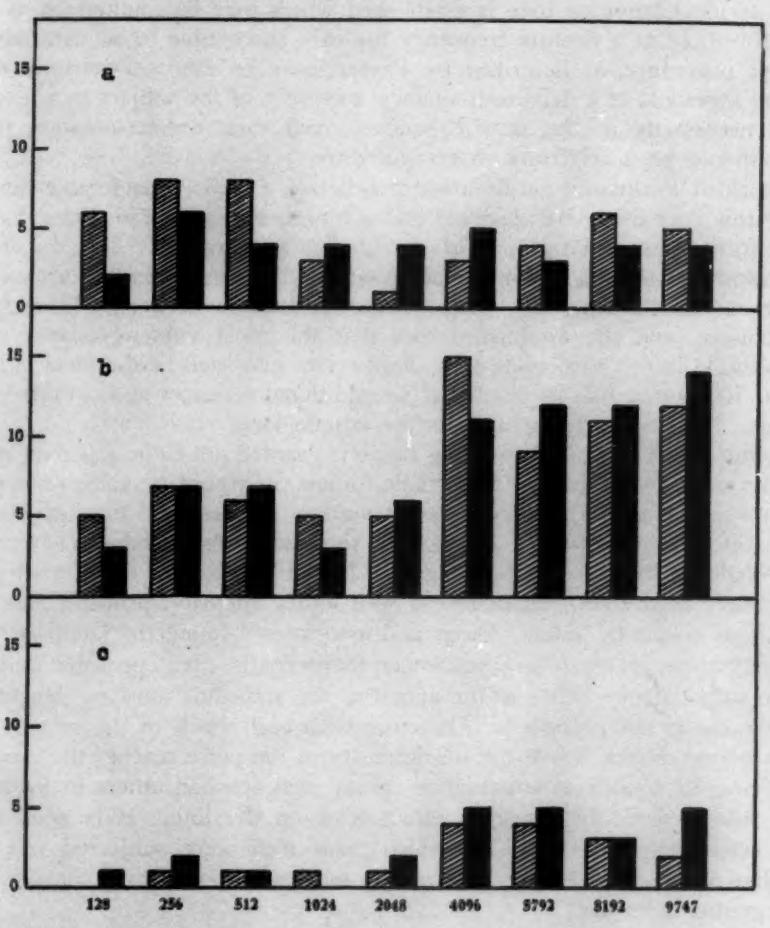

Chart 2.—Number of subjects in whom a rise in threshold was found at each frequency tested: (*a*) after fatiguing, 27 subjects; (*b*) after firing, 31 subjects, and (*c*) after one to two weeks of recovery, 16 subjects.

COMMENT

That there is a definite relation between abnormal auditory fatigue and the irreversible loss of hearing caused by acoustic trauma is apparent from these experimental studies. The greater vulnerability of some ears over others is substantiated. The practical application of this correlation, as previously suggested by Peyser, is that of determining in advance persons who may be unduly susceptible to acoustic trauma. While a means of screening out these susceptible persons would be impractical for use in the armed forces, its employment among men applying for work in noisy industry would reduce compensable auditory loss.

Certainly, it is only by such a method that this ever increasing problem can be coped with. The man who depends on his trade for a livelihood cannot readily be taken away from his work because he shows a lack of resistance to loud sounds. But in the case of the apprentice, the young man who is free to choose his field, one has an opportunity of turning him toward more quiet kinds of work if he proves to be susceptible to occupational deafness. A great service would be done not only to the individual worker but to the industry that would have employed him.

It is a tedious process to take several complete audiograms on each subject. But once an ideal fatiguing tone is established which may be counted on to show a rise in threshold at a definite frequency for ears susceptible to acoustic trauma, the simpler procedure, as described by Peyser, may be used. Determination of the hearing threshold at a definite frequency, exposure of the subject to a fatiguing tone, not necessarily at the same frequency, and then redetermination of the threshold should be a relatively short procedure.

The work of Perlman,[15] as discussed previously, affords considerable information along this very line. He observed that a fatiguing tone of 256 cycles does not give a uniform point of fatigue. This is substantiated by chart 2 *a*. In view of his conclusion that when a rise in threshold occurs it generally reaches a maximum one octave above the fatiguing tone, when the latter is between 512 and 4096 cycles inclusive, and the established fact that the most vulnerable spot in the cochlea is found in the 4096 cycle area, 2048 cycles may well be the ideal fatiguing frequency. It is with this in mind that an additional series of audiograms is now being taken, 2048 cycles being used as the fatigue tone.

While the use of a small arms firing range is granted not to be a perfect laboratory for the exposure of subjects to acoustic trauma, its traumatic value must not be underestimated. The many subjective complaints of deafness and tinnitus when the men leave the range uphold this. So, too, do the many greater changes in threshold curves brought on by exposure to firing than by the fatigue tone of the audiometer.

The nature of the noise of firing, as well as its intensity, probably has much to do with its traumatic value. Davis and associates [16] found the latent period of the tensor tympani reflex to be about fifteen milliseconds. It is probable that practically the same latency exists in the action of the stapedius muscle. During this interval the ear is not protected. Therefore with each crack of the rifle or pistol there is a period during which the full intensity of the noise reaches the inner ear. Besides firing 82 rounds of ammunition, many men coached others in firing, and all were subjected to the firing of other arms on the line. It is readily seen that the actual acoustic trauma to which these men were subjected was much greater than if they had been placed in a room and exposed to a constant noise of much greater intensity.

16. Davis, H.; Derbyshire, A. J.; Kemp, E. H.; Lurie, M. H., and Upton, M.: Function and Histological Changes in the Cochlea of the Guinea Pig Resulting from Prolonged Stimulation, J. Gen. Psychol. **12**:251-276, 1935.

CONCLUSIONS

This series of tests suggests the following conclusions:

The ears of certain persons are more susceptible to acoustic trauma than are those of others. Ears so predisposed are fatigued more readily.

The realization of such a relationship between abnormal auditory fatigue and irreversible traumatic loss of hearing affords a means of determining in advance persons predisposed to traumatic deafness.

Such a means of screening out susceptible persons should be of great value in noisy industry.

An ideal fatiguing tone must be determined that may be depended on to cause fatigue at a definite frequency in these more vulnerable ears.

Persons with impaired hearing are more susceptible to acoustic trauma than are those with normal hearing.

INFLUENCE OF AVULSION OF THE TRIGEMINAL NERVE ON THE HUMAN NOSE

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Although many observations have been carried out on the results of section of the trigeminal nerve in man for tic douloureux, little attention has heretofore been paid to its effect on the nose and the nasal mucosa. This is rather astonishing, as the present day conception of nasal pathology attributes many of its factors to reactions which can only be explained basically as a result of a disturbance of the nerve equilibrium, maintained in the nasal mucosa by its sensory and autonomic innervation. Especially, the wide range of so-called allergic reactions encountered daily with pathologic conditions of the nose should make advisable a morphologic study of noses which have been deprived of their first line of defense, their reaction to pain and irritation. Much of this pathologic change is attributable to mechanical irritation, and it is to be expected that the presence of such irritation, unnoticed by the patient, would have severe effects on the mucosa, as it is known to have in cases of corneal denervation.[1] Here and there the free nerve endings in the epithelium, which indicate the presence of a foreign body, have been eliminated.

On the assumption that some light could be shed on nasal pathologic states, we examined a group of patients who had been operated on for trigeminal neuralgia by Dr. Eric Oldberg and his associates.

Considerable study has been devoted to the neurology of the nasal mucosa, but still a certain divergence of opinion exists. Therefore, before we describe our observations, we wish to review some more recent expressions concerning this important subject, published since Sluder's [2] studies, inasmuch as of late theories have again been advanced explaining systemic and nasal reactions on a neurologic basis and showing the importance of a clear conception of nasal innervation as a basis for their cure.[3]

In our description we shall neglect entirely the fact that the olfactory nerve has its termination in the nose and shall devote our attention only to the sensory and autonomic innervation of the nasal mucosa.

NEUROLOGIC RELATIONS

Macroscopically, the first and second branches of the trigeminal nerve provide all sensory innervation of the nose. Only a small area in the region of the nasal septum receives branches of another, usually neglected, nerve, the terminal nerve.

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1. Ornstein, S.: Untersuchungen über die Sensibilität der Nasenschleimhaut unter normalen und pathologischen Bedingungen, Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **26**:153-175, 1928.

2. Sluder, G.: Nasal Neurology, St. Louis, C. V. Mosby Company, 1927.

3. McLaurin, J. G.: Otorhinologic Origin and Certain Reflexes Caused by Fifth Nerve Irritation, Ann. Otol., Rhin. & Laryng. **50**:469-498, 1941.

TERMINAL NERVE

In spite of the fact that this nerve is fairly well developed in man, knowledge of it is still limited to a few anatomic facts. The terminal nerve originates from the terminal ganglion, which is located medial to the olfactory bulb, just lateral to the crista galli, for the most part within the dura. With three or four rami it enters the nasal cavity, in the anterior portion of the cribriform plate. Its peripheral branches form an interlacing network of nerve fibers along the anterior superior portion of the cartilaginous septum. Many nerve cells are in close proximity to the fibers, which are regarded as sympathetic.[4] Two types of neurons are observed, sensory and motor. Some sensory fibers end in the muscular wall of the anterior cerebral artery, while others terminate freely in the septal mucosa and in the region of Jacobson's organ. They are connected with afferent neurons of the nerve. The innervation of the vomeronasal organ seems to be incidental. Anastomoses have been established with the nasopalatine and the ethmoid branches of the trigeminal nerve, and a sympathetic bundle enters the cribriform plate from the nose, a link with the nasal sympathetic system being thus established.[5]

TRIGEMINAL NERVE AND ITS RELATIONS

Nasociliary Nerve.—The nasociliary nerve of the first trigeminal branch sends a ramus through the anterior ethmoid foramen into the anterior fossa. There this ramus follows the ethmoid sulcus, between the dura and the cribriform plate, into the nasal cavity. Sometimes this nerve is entirely exposed within an ethmoid cell. Its ramification extends over the entire anterior portion of the nasal cavity. The terminal branch passes the nasal bone, extends to the surface and innervates the skin of the nose. The branches for the nasal mucosa have a medial portion for the anterior part of the septum and a lateral portion, which passes the anterior ends of the middle and superior turbinates and spreads in the mucosa of the lateral anterior wall.

Maxillary Nerve.—A few small fibers arising from the infraorbital nerve, via the anterior superior alveolar nerve, reach the inner part of the nose at the anterior portion of the floor of the nasal cavity and communicate with the nasopalatine nerve. A few inner and outer branches around the nasal vestibule arise also from a ramification of the infraorbital nerve. The main innervation of the nose is supplied by the sphenopalatine branches of the maxillary nerve. They contact the sphenopalatine ganglion after a course of a few millimeters.

Sphenopalatine Ganglion.—This triangular or elliptic nerve body lies in the pterygopalatine fossa close to the sphenopalatine foramen. It is enveloped in a thick fibrous membrane and is embedded in adipose tissue. Its connection with the maxillary nerve, to which it lies medially a short distance, consists of one to four fibers, the sphenopalatine nerve. The second of its connections, the vidian nerve, consists of the major superficial petrosal and the great deep petrosal nerve, both encased in a common sheath. An extensive study of the character of the sphenopalatine ganglion has been made by Fenton and Larsell.[6] They stated that embryonically the sphenopalatine ganglion is formed by a migration of cells from the fifth and seventh nerves. In their opinion, the sympathetic character of the

4. Larsell, O.: Study on the Nervus Terminalis in Mammals, J. Comp. Anat. **30**:1-67, 1918.
5. Brookover, C.: Nervus Terminalis in an Infant, J. Comp. Anat. **28**:347-360, 1917.
6. Fenton, R., and Larsell, O.: The Mechanism of Pain Transmission in Certain Types of Otalgia, Ann. Otol., Rhin. & Laryng. **37**:739-761, 1928. Larsell, O.; Burns, J. F., and Fenton, R. A.: Relation of Irritation in the Region of the Paranasal Sinuses to Certain Vasomotor Changes, Arch. Otolaryng. **27**:266-274 (March) 1938. Larsell, O., and Fenton, R. A.: Sympathetic Innervation of the Nose, ibid. **24**:687-695 (Dec.) 1936.

ganglion is manifested by typical multipolar cells encircled by fine fibrillary end nets. Blier [7] and Stewart and Lambert [8] asserted that the sphenopalatine ganglion is a member of the outlying sympathetic ganglions. The afferent fibers of this ganglion come by way of the major superficial petrosal nerve, in which they constitute small myelinated fibers of typical preganglionic character.[6] The postganglionic fibers leave the ganglion with various nerves. Large sensory fibers from the major superficial petrosal nerve pass through the ganglion directly into the palatine nerve, and there are probably other branches. The sphenopalatine ganglion can be regarded as a crossroad for sensory fibers from the facial ganglion (ganglion geniculi) and from the petrous ganglion of the glossopharyngeal nerve (by way of Jacobson's nerve); for cranial autonomic fibers, secretory, from the greater superficial petrosal and maxillary nerves; for thoracicolumbar fibers, secretory, from the superior cervical sympathetic ganglion; for vasoconstrictor fibers from the superior cervical sympathetic ganglion; for vasodilator fibers from the greater superficial petrosal nerve and, finally, for sensory fibers, of unknown origin, which pass through the ganglion.[6] That these afferent fibers may be sensory is supported by Fenton's [9] experiments, in which he noted vasodilatation in the ear after faradic stimulation of the sphenopalatine region in rabbits in which the trigeminal nerve had been cut.

Vidian Nerve.—The major superficial petrosal nerve originates in the geniculate ganglion of the facial nerve. It reaches its sulcus on the pyramidal surface after arising from the facial hiatus, perforates the sphenopetrous synchondrosis lateral to the carotid artery and associates here with the great deep petrosal nerve, which arises at this point from the nerve plexus around the artery. It passes as the vidian nerve through the vidian canal to reach the sphenopalatine ganglion. Vogel [10] expressed the opinion that the vidian nerve is interrupted as an entity after entering the ganglion. A number of fibers arise from the vidian nerve directly, without entering the sphenopalatine ganglion, and innervate the mucosa of the sphenoid sinus and of the pharynx. A direct anastomosis also exists between the greater superficial petrosal nerve and the tympanic plexus. The experiments of Jung, Tagand and Chavanne [11] have shown that some secretory fibers for the nasal mucosa run along with the maxillary nerve, but the main supply comes from the vidian nerve. Stimulation of the vidian nerve results in copious secretion, while resection of the nerve diminishes the secretion.

Major Superficial Petrosal Nerve.—The major superficial petrosal nerve is regarded as parasympathetic, bringing secretory fibers for the nose,[12] the preganglionic fibers arising from the medullary bulb.[13] But there is no doubt that afferent fibers are contained in the major superficial petrosal nerve, and also in the chorda tympani.[6] They are largely myelinated, pass through the sphenopalatine ganglion without interruption and reach the cells of Yagita in the geniculate ganglion, which are of unipolar sensory type. Larsell and Burns [13] called them

7. Blier, Z.: Physiology of the Sphenopalatine Ganglion, Am. J. Physiol. **93**:398-406, 1930.

8. Stewart, D., and Lambert, V.: The Sphenopalatine Ganglion, J. Laryng. & Otol. **45**: 753-771, 1930.

9. Fenton, R. A.: Some Observations on Nasal Neurology, Ann. Otol., Rhin. & Laryng. **50**:492-498, 1941.

10. Vogel, K.: Die Beziehungen des Ganglion sphenopalatinum zu den von ihm ausgehenden Nervenstämmen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **25**:485-502, 1930.

11. Jung, L.; Tagand, R., and Chavanne, F.: Sur l'innervation excitosécrétoire de la muqueuse nasale, Compt. rend. Soc. de biol. **95**:835-837, 1926.

12. Rowbotham, G. F.: Observations on the Effects of Trigeminal Denervation, Brain **62**:364-380, 1939.

13. Larsell, O., and Burns, E. M.: Some Aspects of Certain Cranial Nerves, Ann. Otol., Rhin. & Laryng. **40**:661-669, 1931.

palatine branches of the facial nerve. Ruskin[14] and Rowbotham[12] attributed gustatory qualities to the sensory part of the greater superficial petrosal nerve. Sappey, Pitres and Testut[15] called it a motor nerve which passes through the ganglion to supply the palatine muscles. Alexander[15] observed the sensory fibers in the greater superficial petrosal nerve, extending from the trigeminal to the peripheral position of the facial nerve. Stewart and Lambert[8] asserted that the sympathetic part of the greater superficial petrosal nerve is the only true root of the sphenopalatine ganglion. In the opinion of French authors, cited by Bertein,[16] parasympathetic fibers arising from the seventh and the ninth nerve reach the vidian nerve from Jacobson's nerve via the greater superficial petrosal and deep petrosal nerves and have their cell station in the sphenopalatine ganglion. Electrical stimulation of the peripheral part of the greater superficial petrosal nerve causes dilatation of blood vessels and glandular secretion, the same result as that obtained with stimulation of the ganglion and its branches.[17]

Deep Petrosal Nerve.—The deep petrosal nerve consists of unmyelinated fibers, postganglionic sympathetic, from the superior cervical sympathetic ganglion, but it contains also a considerable number of large and medium-sized myelinated fibers, which appear to derive from other sources, perhaps the petrous ganglion, and which reach the deep petrosal nerve by way of Jacobson's nerve and the small petrosal nerve. Some myelinated sensory fibers coming down the major petrosal nerve appear to be shunted around the carotid plexus into connection with the glossopharyngeal nerve, and through it with the vagus nerve.[16] It is generally agreed that the sympathetic fibers of the deep petrosal nerve pass through the sphenopalatine ganglion without interruption. French authors have expressed a different opinion. They asserted that the great deep petrosal nerve is parasympathetic, its fibers coming from the glossopharyngeal nerve via Jacobson's nerve, while the sympathetic fibers arise from the plexus of the cavernous sinus. This view is most explicitly stated in Bertein's[16] study. The sympathetic fibers from the superior cervical sympathetic ganglion reach the nasal mucosa in the adventitia of the blood vessels, mainly the external carotid artery. The fibers from the great deep petrosal nerve accompanying the internal carotid artery form a plexus and pass through the sphenopalatine ganglion without interruption to follow the other nerves.

Branches of the Sphenopalatine Ganglion.—A connection exists between the otic and the sphenopalatine ganglion. Through the sphenopalatine foramen a group of superior nasopalatine nerves reach the mucosa, arising directly from the ganglion. The medial bundle, Scarpa's nasopalatine nerve, reaches the nasal septum, on which it remains in its course anterior and inferior until it reaches and passes through the incisive canal to the hard palate. The lateral branches, six to ten fine filaments, spread in the region of the posterior portion of the middle and inferior turbinates, the posterior ethmoid cell and the posterior choana. One branch reaches the region of the superior turbinate. From the main nerve stem, the palatine nerve, arising from the sphenopalatine ganglion, only a branch of the anterior palatine nerve reaches the nose, as the lateral inferior nasal nerve. It arises from the anterior palatine nerve within its course in the pterygopalatine canal and extends to the inferior turbinate. All the other palatine branches have

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16. Bertein, R.: Essai anatomie sur le systémé sympathétique de la muqueuse pituitaire, Arch. internat. de laryng. **4**:520-548, 1925.
17. Jung, L.; Tagand, R., and Chavanne, F.: Innervation fréno-sécrétoire de la muqueuse nasale, Compt. rend. Soc. de biol. **96**:1315-1316, 1927.

no contact with the nasal mucosa. The palatine nerve itself contains fibers which arise from the maxillary nerve directly and reach it without passing through the sphenopalatine ganglion. Other branches from the sphenopalatine ganglion form a direct connection with the orbit. There exist a posterior and an anterior ramification of these filaments. The anterior branches reach the orbit by way of the inferior fissure, enter the nasal cavity through the posterior ethmoid opening of the papyraceous plate and supply all the ethmoid cells and the sphenoid sinus, and sometimes also the maxillary sinus. Other branches contact ocular muscles and form a root of the ciliary ganglion. The posterior orbital ramification of the sphenopalatine ganglion contains fibers which have been observed to enter the optic nerve. Another filament is always seen which leads to the sympathetic plexus of the cavernous sinus; occasionally a communication with Gasser's ganglion is observed, as well as branches to the extraocular muscles and to the smooth muscles of the orbit.[18] The ganglion supplies most of the innervation for the pterygopalatine vessels, with a few fibers arising from the trigeminal nerve directly. The sphenopalatine nerves enter the ganglion only with a fraction of their fibers. The greater part pass anterior and lateral to the ganglion and form, together with fibers arising from the ganglion, the palatine nerves. Among them, the palatinus major consists mostly of fibers arising from the maxillary nerve directly; only a few posterior fibers arise from the ganglion. The posterior superior nasal nerve, Scarpa's nerve and the branches for the posterior region of the nose and pharynx arise directly from the ganglion alone. But fibers for the middle turbinate, which arise from the inferior pole of the ganglion, as well as fibers for the middle meatus and the inferior turbinate, arising from the anterior palatine nerve, contain uninterrupted fibers from the maxillary nerve. The fibers entering the orbit do not contain any direct fibers from the maxillary nerve.[10] The purely postganglionic character of the posterior nasal and palatine nerves is being widely contradicted, as it is recognized that the greater portion of nerve fibers pass the ganglion without interruption.

AUTONOMIC NERVOUS SYSTEM

Sympathetic System.—According to Christensen,[19] the nerves for the nasal mucosa and nasal sinuses contain a number of unmyelinated fibers, which are postganglionic sympathetic. Unmyelinated fibers observed in the maxillary and vidian nerves arise from the cervical sympathetic plexus and pass through the sphenopalatine ganglion without interruption. A few originate in the upper thoracic ganglions, disappearing when the superior cervical sympathetic ganglion is extirpated.[20] The rest of the fibers, mostly small and large myelinated fibers and a few unmyelinated fibers, are probably all sensory and have their origin in the semilunar ganglion. The preganglionic sympathetic fibers, vasoconstrictor for the nasal mucosa, are observed in the cervical sympathetic plexus. Their synapse occurs in the superior cervical ganglion, and the postganglionic fibers run in the plexus round the internal carotid artery.[7] A major portion reaches the posterior nasal nerve; a minor part forms the deep petrosal nerve, traverses the sphenopalatine ganglion and reaches the mucosa in this way. Only Schaeffer, cited by

18. Tanaka, T.: Ganglion sphenopalatinum des Menschen, Arb. a. d. dritten Abt. d. anat. Inst. d. kaiserl. Univ. Kyoto (ser. A, pt. 3), 1932, pp. 91-115.

19. Christensen, K.: Innervation of the Mucous Membrane of the Nose and Paranasal Sinuses with Particular Reference to the Sensory Fibres, Tr. Am. Laryng., Rhin. & Otol. Soc. **42**:363-370, 1936.

20. Kure, K., and Sakurasawa, S. F.: Ueber die parasympathetischen Fasern für das Ganglion sphenopalatinum usw., Ztschr. f. Zellforsch. u. mikr. Anat. **9**:245-255, 1929. Larsell and Burns.[13]

Stewart and Lambert,[8] contended that a few sympathetic fibers may have their cell station in the sphenopalatine ganglion. The French school observed a bundle separate from the carotid sympathetic plexus in the cavernous sinus and enter the gasserian ganglion to follow the distribution of its three branches, a course which gives them a vegetative character. So also the nasal branches of the trigeminal nerve contain vegetative fibers. These carotid fibers are most likely secretory. All these fibers are observed in the mucosa and cannot be distinguished from the purely trigeminal terminations. They are noted around glands and vessel sheaths and have no connection with the branches of the olfactory nerve. The cervical sympathetic plexus exercises a restraining action on the nasal secretion, but occasionally has a vasodilator effect. As to the other functions of the sympathetic system, Burkart [21] stated that it has definite calorigenic properties in the nasal mucosa. Referred pains are often attributed to the sympathetic nervous system, and Peet [22] expressed the belief that vagus fibers may possibly be incorporated in the sympathetic portion of the facial nerve. This association may be a link for transmission of painful vascular sensations, and only in this gross sense can the sympathetic system be regarded as sensory.

Parasympathetic System.—The presence of parasympathetic fibers in the trigeminal nerve was established by Moteki.[23] He observed a group of small ganglion cells in the gasserian ganglion which remained unchanged after section of the posterior root, while degeneration of similar cells took place when peripheral branches were severed. These cells must be regarded as constituting a parasympathetic cell station. According to Kure and Sakurasawa [20] the sphenopalatine ganglion receives parasympathetic fibers by way of the vidian and the maxillary nerve. About one third of the small myelinated fibers of the maxillary nerve end in the sphenopalatine ganglion and must be regarded as preganglionic parasympathetic. The greater number of parasympathetic preganglionic fibers is supplied to the nose by way of the greater superficial petrosal nerve, with all the cell stations in the sphenopalatine ganglion.

Comment.—Thus the nasal nerves contain sensory, parasympathetic and sympathetic fibers. Both nervous systems are mixed and can produce vasodilatation and vasoconstriction. Section of the sympathetic fibers will produce vasodilatation in the conjunctiva, iris, ear, tongue and epiglottis and vasoconstriction in the retina, lips, cheek, nasal mucosa and glands. No trophic influence is present, as section of the sympathetic fibers results merely in inhibition of secretory fibers. Stimulation of the sympathetic fibers produces abundant, thick secretion, rich in mucin, from the glandular epithelium, while parasympathetic irritation of the nasal mucosa is followed by serous secretion subsequent to vasodilatation, which cannot be regarded as a secretory effect, but is to be considered only as transudation.[16]

Disturbance of this equilibrium in the nose maintained by the autonomic nervous system may show as vasomotor congestion and signify a decrease in the nasal sensory threshold.[24] Pharmacologic studies led Sternberg [25] to the following

21. Burkart, A.: Untersuchungen über den Einfluss des Nervus sympatheticus auf die Temperatur der Nasen- und Mundschleimhaut, Ztschr. f. Biol. **97**:464-473, 1936.

22. Peet, M. M.: The Role of the Sympathetic Nervous System in Painful Diseases of the Face, Arch. Neurol. & Psychiat. **22**:313-321 (Aug.) 1929.

23. Moteki, S.: Parasympathetische Fasern im N. trigeminus und trophische Innervation derselben, Klin. Wchnschr. **18**:25, 1939.

24. Vernet, M.: Du rôle primordial de la sensibilitée organique dans la pathologie des voies respiratoires supérieures, en particulier par rapport aux manifestations du sympathique, Acta oto-laryng. **12**:120-131, 1928.

25. Sternberg, H.: Pharmakologische Reaktionen am Schwellkörper und subepithelialen Kapillarnetz der Nasenschleimhaut, Monatschr. f. Ohrenh. **63**:390-395, 1929.

conclusions: Irritation of the vidian nerve, sphenopalatine ganglion and posterior nasal nerve always results in dilatation of the vessels of the nasal mucosa, as vasodilators prevail in these nerves. When the dilators are eliminated by painting the sphenopalatine ganglion with nicotinic acid or by application of cocaine to the nasal mucosa *"als Erfolgsorgan,"* vasoconstriction results from irritation of these nerves. But this reaction is not as effective as irritation of the cervical sympathetic fibers. Therefore vasoconstrictor fibers must also reach the nasal vessels from the cervical sympathetic ganglion by other ways. Differentiation has to be made between the vessels of the cavernous bodies and the subepithelial capillaries. The two systems may react alike or may oppose each other, as is seen when the mucosa of a shrunken nose is reddened or the mucosa of a swollen nose appears pale. The subepithelial capillaries do not seem to be influenced by irritation of the cervical sympathetic fibers.

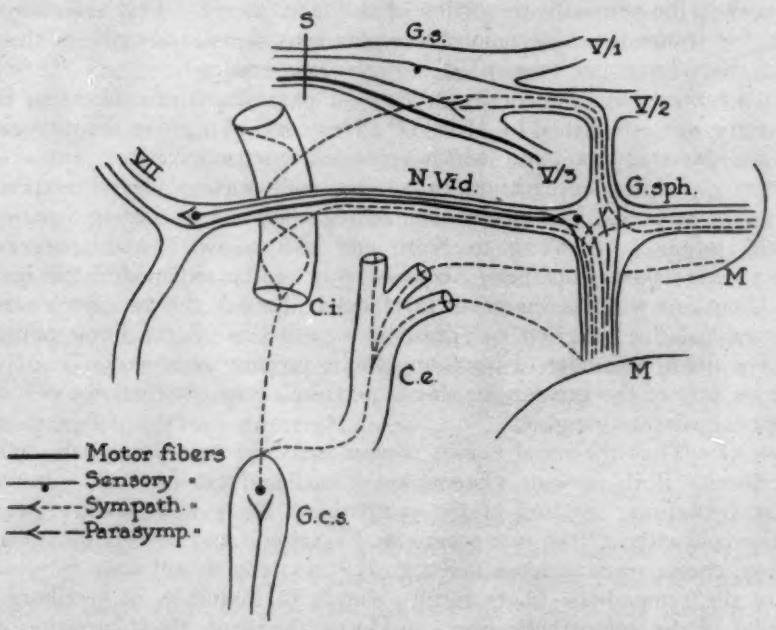

Diagram showing relations of the trigeminal nerve. Here, *G. s.* indicates the ganglion semilunare; *G. c. s.*, ganglion cervicale superius; *G. sph.*, ganglion sphenopalatinum; *C. i.*, carotis interna; *M*, mucosa nasalis; *V1, V2* and *V3*, first, second and third branches of the trigeminal nerve; *VII*, nervus facialis; *N.Vid.*, nervus vidianus; *C.e.*, carotis externa, and *S*, place of sectioning.

HISTOLOGIC FEATURES

The average nasal mucosa contains a certain relative amount of connective tissue, lymphocytes, other lymphoid cells, eosinophils, blood vessels, cavernous vessels, glands and lymphatic and distended interstitial spaces.[26] Normally round cells are accumulated below the basal membrane and in the neighborhood of glands.[27] In the submucosa and the tunica propria nerve fibers, which are mostly unmyelinated, are observed;[28] they arborize around blood vessels, cavernous

26. Finck, H. P.: Tissue Changes in the Nasal Mucosa, Laryngoscope **37**:783-797, 1927.

27. Hett, J.: Zur Histologie der Nasenschleimhaut, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **143**:406-412, 1937.

28. Kadanoff, D.: Beiträge zur Kenntnis der intraepithelialen Nerven des Menschen: III. Die Nerven im Epithel der Nasenschleimhaut, Ztschr. f. Zellforsch. u. mikr. Anat. **6**:342-347, 1927.

spaces and glands. There is no subepithelial plexus, but fibers ascend obliquely or rectangularly and end free in the epithelium. Nerves observed around glandular ostiums are probably sensory.

REPORT OF CASES

From the material of the neurosurgical department of the University of Illinois College of Medicine, 10 cases were selected in which an avulsion of the trigeminal nerve had been performed and the nasal condition examined. The history of nasal complaints was taken; the nose was thoroughly examined; sensibility was tested, and in each case a small piece of mucosa was removed from either the middle or the inferior turbinate body on each side. The removed piece was fixed in formaldehyde and, after being sectioned, was stained with hematoxylin and eosin. We wish to present the histories in these cases in order, according to the time which elapsed between the day of the trigeminal avulsion and the nasal examination.

CASE 1.—The longest interval was observed in the case of R. S., a man aged 68, whose trigeminal neuralgia had appeared at the age of 21. He had avulsion of the second and the third branch of the right trigeminal nerve in 1902 and 1906 respectively and of the left trigeminal in 1926 and 1939, the last operation finally being followed by relief of his complaints. He had had complete anesthesia of the right side of the face since 1906 and of the left side since 1939. He did not give any history of nasal complaints and had only one or two colds a year, with little discharge. There was no sneezing or stuffiness of the nose. He noticed that the right side of his nose seemed a little drier than the left. On examination, the skin of the surface showed equal degrees of hypesthesia on the two sides, while the right side of the nose evidenced less sensibility to touch than the left. Nasal inspection revealed a slightly soggy, waterlogged, pale mucosa on the left side, with increase of secretion. The sinuses were clear. After application of cocaine the right middle turbinate was paler than the left. Biopsy was made of tissue from both middle turbinates.

Left Middle Turbinate (side of the more recent operation).—The epithelium was partly squamous and partly columnar, with mucoid degeneration, and contained some crypts, with nonproducing columnar epithelium. Migration of polymorphonuclear leukocytes into the epithelium was present. Some papillae contained blood vessels in the squamous epithelium. The nasal membrane was partly thickened. The stroma showed slightly edematous connective tissue, somewhat infiltrated with polymorphonuclear leukocytes, lymphocytes and some eosinophils. The vessels were dilated and empty, with no evidence of perivascular infiltration. Quiescent, active and hyperactive mucous glands and many serous glands were seen.

Right Middle Turbinate.—The epithelium was columnar, with mucoid degeneration, and contained deep crypts. The basal membrane was heavy and enormously thickened in some parts. The stroma was edematous, with little evidence of infiltration, and there were few eosinophils. The vessels were dilated and enormously filled, with no perivascular infiltration. There were relatively few glands, and all were inactive (as a consequence of the edema). One glandular group showed signs of degeneration; periglandular infiltration was lacking; the tubules were at a greater distance from each other than normal and had no lumen, but their cells showed vacuoles, and all the tubules were surrounded by hyalinized connective tissue.

CASE 2.—I. V., a woman aged 73, had had an avulsion of the left trigeminal nerve seven years ago, followed by enucleation of the left eye for a perforating ulcer. A hornifying squamous cell carcinoma which developed in the left vestibular region of the nose was successfully treated with radium. Her nasal history, as far as could be elicited, was entirely without significance. Examination revealed anesthesia of the left side of the face and nose, where the sense of temperature was also lacking and a very hot application was noted as cold. The mucosa of the left side was paler and presented a more shrunken appearance than the right. A speciment for biopsy could be taken without anesthesia. Bleeding following removal of the tissue was about equal on the two sides and was readily controlled.

Left Inferior Turbinate.—The epithelium was of high columnar type, with no evidence of mucoid degeneration. The basal membrane was partly thickened. The connective tissue was edematous, without infiltration. Quiescent, active and hyperactive glands were noted. The vessels appeared unchanged.

Right Inferior Turbinate.—The epithelium was of high columnar type. The nasal membrane was partly thickened, and the vessels were normal. Loose connective tissue was present, without infiltration or edema. Quiescent and active glands were seen.

CASE 3.—M. P., a woman aged 45, had had partial avulsion of the posterior root, involving the second branch, of the right trigeminal nerve three years and four months ago, followed by disappearance of pain in the right cheek. She still distinguished cold and heat on her cheek. Her history revealed no nasal difficulty. She did not sneeze except occasionally when she combed her hair. She had had no colds for the last six years, no discharge from the nose and no stuffiness. The nose felt rather dry, but she was not able to tell which side was more so. The right side of the nose seemed to be more sensitive to cold air than the left. The sinuses were clear. The skin of the right side of the face was numb, and the nose was a little wider on the right side. There was no macroscopic difference in the mucosa of the two sides. A few dry crusts were present on the right side; a little mucus was noted on the left. The color of the mucosa was normal. There was pronounced hypesthesia on the right side. Both inferior turbinates shrank equally well with cocaine. Specimens were taken from both turbinates for biopsy.

Right Inferior Turbinate.—The epithelium was transitional in type and showed no mucoid degeneration. The basal membrane was thickened. The stroma was loose, nonedematous connective tissue, with slight infiltration, no eosinophils and normal vessels. No glands were observed.

Left Inferior Turbinate.—The epithelium was necrotic; the basal membrane was thickened, tough and nonedematous, and the connective tissue was not infiltrated. Dilated vessels, without perivascular infiltration were observed. The glands were partly necrotic, with no signs of activity. It was difficult to account for this condition in a supposedly normal nose.

CASE 4.—C. S., a man aged 65, had had a resection of the posterior root of the left trigeminal nerve two years ago, but as pain in the eye recurred, a second operation was performed six days prior to the nasal examination. He reported having one to two colds a year and sometimes a stuffy nose and a slight mucoid discharge. The skin of the left side of the face was anesthetic. There were marked deviation of the septum to the left and loss of sensibility for pain on the left side of the nose. The mucosa on the left side was decidedly paler than that on the right, and the membrane appeared soggy. There was no discharge, but crusting was present. The sinuses were clear. A biopsy specimen was taken from the middle turbinates.

Left Middle Turbinate.—The squamous epithelium was without mucoid degeneration; no crypt formation was seen. The basal membrane was thickened; the stroma was not infiltrated, and there was no edema. Arterioles and large veins, without perivascular infiltration, were present. The glands were observed to be active or hyperactive, and some were about to rupture.

Right Middle Turbinate.—The epithelium was partly stratified columnar and partly squamous in type. There was little mucoid degeneration of the columnar cells, but on the surface of the epithelium several completely mucoid excretory ducts were observed. A layer of mucus covered the epithelium. Few crypts were seen, and the basal membrane was partly thickened. There was a normal stroma of connective tissue, with few eosinophils, very few lymphocytes and no edema; the vessels were slightly dilated and empty. No perivascular infiltration was present. Quiescent, active, hyperactive and rupturing glands were in evidence. Numerous glands and considerable periglandular infiltration were observed.

CASE 5.—A. P., a woman aged 39, had had section of the left trigeminal nerve sixteen months previously, with relief. She admitted having two or three colds every winter. Sometimes the right side of the nose became stuffy. She expressed the belief that she had had fewer colds since the operation. There was little discharge, the left side of the nose feeling drier and there being no sensation on inhalation. The left side of the face was anesthetic. There was deviation of the septum to the right; the mucosa appeared alike on the two sides of the nose, with no difference in color or moisture. There was notable lack of sensibility on the left side. Touch was noted, but not pain. The antrums appeared clouded on transillumination. The mucosa evidenced good shrinkage on both sides. Biopsy of material taken from the inferior turbinates revealed the following changes:

Left Inferior Turbinate.—The epithelium was of squamous and transitional type, with a thickened basement membrane. The connective tissue was nonedematous and not infiltrated. The vessels appeared normal; the glands were exhausted and evidenced but little activity.

Right Inferior Turbinate.—The epithelium was columnar, with mucoid degeneration. The basement membrane was partly thickened. The stroma appeared edematous and was but slightly infiltrated. The glands were sparse and evidenced no activity.

CASE 6.—M. C., a woman aged 73, had had a successful avulsion of the second and third branches of the right trigeminal nerve thirteen months ago. She did not remember any particular nasal complaint except that pus came from her nose after colds and there was a slight postnasal drip; she had rare attacks of sneezing. The right side of the face was numb.

The nose was narrow, with no apparent difference in the mucosa or the amount of discharge on the two sides. Sensibility on the right side was reduced. The sinuses appeared clear. Biopsy of specimens from the middle turbinates evidenced the following changes:

Right Middle Turbinate.—The epithelium was transitional and columnar, with slight mucoid degeneration. The basement membrane was partially thickened. The stroma was rich in nuclei; it was nonedematous and showed little infiltration. The vessels appeared normal. Many glands were observed, which were quiescent, active or hyperactive. Few leukocytes were present.

Left Middle Turbinate.—The epithelium was of transitional type, with partial mucoid degeneration, and in some areas there were highly vascularized papillae, but no crypts. The basement membrane was greatly thickened. The stroma was of loose, partly hyalinized connective tissue, with little infiltration, no edema and a few eosinophils. The vessels were dilated, with slight perivascular infiltration. There was good periglandular infiltration; the glands were quiescent or exhausted, with little production of mucus.

Case 7.—G. B., a man aged 55, had had an avulsion of the sensory root of the left trigeminal nerve a year ago, with elimination of pain. He did not recall having had any nasal trouble. Immediately after the operation he had a severe sneezing spell. There was a slight postnasal drip. The left side of the face was paler than the right, and the right middle turbinate showed slight polypoid degeneration. The sinuses appeared clear. Biopsy of tissue from the middle turbinate revealed the following changes: The left (side of operation) and the right middle turbinate were essentially alike. The epithelium was partly of squamous type and partly of columnar type with slight mucoid degeneration; there were no crypts, and the basal membrane was normal. The stroma was sparsely infiltrated, with few eosinophils and some pigment cells. There was no subepithelial layer and no edema. The vessels were dilated, without filling or perivascular infiltration. Glands were numerous, the major portion being serous, and the periglandular infiltration was good. The mucous glands as a rule were hyperactive; some had ruptured; very few were exhausted. There were few pyknocytes.

Case 8.—F. C., a woman aged 35, had had avulsion of the sensory root of the right trigeminal nerve seven months ago, after which she suffered from Bell's palsy. Her nasal history was without significance, with only rare colds, no discharge from the nose and no sneezing. The right side of the face and nose felt dry and numb. The nasal mucosa showed no change and no disturbance in secretion. In this case there did not seem to be a major difference in sensibility, except in the region of the right septal tubercle. The sinuses were clear. Biopsy specimens revealed that the right (side of operation) and the left middle turbinate were essentially alike: The epithelium was of squamous type, with high vascularized papillae. The stroma was not infiltrated or edematous. The basal membrane was thin. The vessels were dilated, with no perivascular infiltration. No glands were encountered.

Case 9.—R. W., a woman aged 70, had had a section of the second and third posterior roots of the right trigeminal nerve six months before she was seen, with improvmeent. The patient had only rare colds, but noticed a watery discharge from the nose when the temperature changed. Sometimes she had parosmia. Since the operation she noticed the cold air more in her right nostril. The right side of the face was anesthetic. The nose was entirely normal, with no discharge. Sensibility was reduced on the right side. The sinuses were clear. Biopsy of tissue from the inferior turbinates revealed the following changes:

Right Inferior Turbinate.—The epithelium was partly missing and partly squamous and was penetrated by excretory canals, which were filled with mucus. The basal membrane was thickened. The stroma was not infiltrated, the connective tissue being partly edematous and the vessels dilated and well filled, with no perivascular infiltration. Glands were many and were quiescent, active, hyperactive or ruptured. The serous glands were also partly dilated and contained thickened secretion.

Left Inferior Turbinate.—The epithelium was partly missing and partly of low squamous type, with shallow crypts. The basal membrane was very much thickened. The stroma showed no infiltration, little edema, no eosinophils, dilated vessels and no perivascular infiltration. Glands were plentiful; they were quiescent or exhausted, with no production of mucus.

Case 10.—J. S., a woman aged 54, had been relieved of trigeminal neuralgia on the left side by section of the second and third posterior roots five months before she was seen. She had had many colds and influenza nearly every winter; otherwise, her nose was normal, with no discharge and no sneezing. The left side of her face was anesthetic in the distribution of the second and third branches. The nose was wide; there was no difference in the appearance of the mucous membrane on the two sides. No marked discharge was present. Sensibility on the left side was reduced. The sinuses were clear. Biopsy of tissue from the middle turbinates revealed the following changes:

Left Middle Turbinate.—The epithelium was columnar and transitional, with no mucoid degeneration. The basal membrane was thickened. The stroma contained many nucleoli; it was not infiltrated. Only subepithelially was there a thin layer of connective tissue which was edematous and infiltrated. No eosinophils were present. The vessels were normal. The glands were active, hyperactive, ruptured or exhausted.

Right Middle Turbinate.—Transitional epithelium, which did not show mucoid degeneration, and columnar epithelium, with signs of mucoid degeneration, were present. The basal membrane was normal and in some spots thickened. The stroma consisted of loose, nonedematous connective tissue, with little infiltration. There were no eosinophils; the vessels were normal. There were many active, hyperactive and quiescent glands and many serous glands with retention of secretion. Oncocytes were noted.

SUMMARY OF STUDY

The 10 patients who were examined ranged in age from 35 to 73 years. Three were men and 7 women. The time which had elapsed from the nerve avulsion to the nasal examination (the latter taking place in April and May 1941) varied from five months to thirty-nine years, except in a case in which a second operation had been done on the same side six days prior to the nasal examination, the primary operation having been performed two years previously.

The intervals between the two operations and the nasal examination in case 1 were thirty-nine years and two years respectively; in case 2 the lapse was seven years; in case 3, three years and four months; in case 4, two years; in case 5, sixteen months; in case 6, thirteen months; in case 7, twelve months; in case 8, seven months; in case 9, six months, and in case 10, five months.

The duration of the neuralgic pain, which also should be borne in mind, was ten months to several years prior to the operation. The general health of all the patients was fair. When the nasal histories of these patients, all inhabitants of Chicago, are compared, the lack of any major complaint is remarkable. The first question, as to colds, revealed that most of the patients suffered from not more than two a year, which can be regarded as within normal limits. Two patients (cases 3 and 5) even stated that since the operation their susceptibility to colds seemed to have diminished (in case 6 there had been no colds for six years; in case 10, frequent colds).

The next question relative to attacks of sneezing, was answered in the negative by 8 patients; naturally, physiologic sneezing was present, and no difference in this respect was noted after operation. One patient (case 3) reported she sneezed when combing her hair; another (case 7) had a severe spell of sneezing immediately after the operation, but there was no previous or further complaint. Temporary stuffiness of the nose was noted by 1 patient (case 5), who also had a deviated septum. Five patients stated that the nose was dry, or at least there was so little discharge as not to be noteworthy. Three patients noted a slight postnasal drip, and 2, a difference in the nasal secretion, the side of operation being dry. Two patients felt cold air more strongly on the side of operation. One patient gave a history of parosmia; the others had a normal sense of smell. One patient noted a watery discharge at the least drop in temperature. For all patients the primary objective of the operation had been achieved. The patients were free of complaints, and hypesthesia or complete anesthesia of the involved area of the face was present. None of them showed any major deformity of the nose except for deviation of the septum. There was no conformity in the shape of the nasal cavity, some noses being wide and some narrow.

In examination of the nasal mucosa an attempt was made to compare the appearance, color, sensibility to touch and pain and discrimination of heat and cold on the two sides. The effects of shrinkage were observed. Six patients did

not show any changes in the normal appearance of the mucosa. In 2 the membrane on the side of operation was waterlogged and soggy, especially over the turbinates. In 1 patient the mucosa gave the impression of being shrunken. A paler mucosa was seen on the side of operation than on the normal side in 4 patients. One evidenced slight polypoid degeneration of the middle turbinate on the normal side. Mucoid secretion was seen to be increased on the left side in the first patient. Minor formation of crusts on the side of the operation was noted in 2 patients.

Specimens for histologic examination were obtained from the middle and inferior turbinates of 5 patients. No abnormal reaction to shrinkage with ephedrine or cocaine was noted; also, bleeding after removal of the biopsy specimen seemed normal. In 4 patients columnar epithelium, combined with transitional and squamous epithelium, was present on the side of the nerve section. The columnar epithelium evidenced mucoid degeneration. In 4 other patients squamous epithelium was noted, being combined in 1 instance with transitional epithelium but showing no mucoid degeneration. In 1 patient only high columnar epithelium without mucoid degeneration, and in another a layer of transitional epithelium with slight mucoid degeneration, was observed. The epithelium on the "healthy" side was columnar, with evidence of mucoid degeneration, in 6 patients, being combined with squamous epithelium in 2 of them. A squamous cell surface with crypt formation was observed in 2 patients; the surface covering was of transitional type in 1 patient and necrotic in another. A difference in the epithelium of the two sides of the nose was noted in patients 1 and 5, in whom only columnar epithelium was seen in the nondenervated side of the nose, while the other side showed squamous and transitional epithelium. The most noteworthy difference encountered in comparison of the epithelial covering of the two sides was the smaller amount of mucoid degeneration on the denervated side. The basement membrane was thickened in 8 patients; in 1 it was normal and in 1 thin. There was no major difference between the two sides of the nose in this respect. The stroma of the mucosa revealed more or less loose connective tissue with slight grades of edema in 3 patients, especially noticeable in the side of operation in patient 2. Little or no infiltration with leukocytes was evidenced, the scarcity of the eosinophils being particularly noteworthy. Only in patient 10 was there a difference between the two sides of the nose, with subepithelial edema and infiltration, and in case 7 the intact side showed hyalinization. The blood vessels were unchanged, being partly dilated and filled and partly empty. There was no perivascular infiltration. There was no remarkable difference in the two sides in this respect. Pyknocytes and oncocytes were observed. The number and activity of the glands were more or less equal on the two sides. Only in patient 1 were inactivity and degeneration noted on the right side, on which operation had been performed many years ago, while the other side was normal. In all the other patients the amount and activity varied from sparseness and exhaustion of the serous and mucous glands to a multiplicity of changes—exhaustion, quiescence, activity, hyperactivity and rupture —all observed in the same specimen. Fair to good periglandular infiltration was present.

Eight patients displayed hypesthesia or complete anesthesia to touch of the nasal mucosa on the side of operation. In patient 1 the right side, on which operation had been done years ago, was less sensitive than the left, on which a recent operation had been performed. One patient was not certain about a difference. Patient 2 could not discern heat and cold on the side of operation.

COMMENT

Comparatively little study has been devoted to the influence on the nasal mucosa of experimental section of the trigeminal nerve. Generalized statements concerning nasal changes have been made on the basis of the results anticipated from nerve sections in other parts of the body, but these claims have not always been substantiated by facts. Bernard (cited by Bertein [16]) noted ulceration in the nose after section of the trigeminal nerve, but it was supposedly inconstant. Damage to the cornea and eye is too well known to require discussion, but the fact that the face is better protected from trauma than the rest of the body seems to be the main reason that the disturbance of sensory innervation in this region only rarely results in ulceration (Karnosh and Scherb [29]). When we compare our histologic observations with Roessle's [30] description of the so-called physiologic inflammation of the nasal mucosa, we must admit that in none of our cases were there any major changes which could not be noted in any person of the same age living in the circumstances of a present day city. Roessle's description of a low level of physiologic inflammation goes, in some respects, even a little further than the pictures which we encountered. He spoke of the presence of a certain minimum of large and small mononuclear round cells in the mucosa, with occasional subepithelial aggregations of lymphoid cells, forming a lymph nodule; occasional plasma cells or eosinophils were encountered. In our cases there was an average amount of connective tissue. No excessive localized or interstitial edema, no changes in the vessels and glands as a rule and no polymorphonuclear leukocytes in the stroma were apparent. Migratory cells could be seen among the epithelial elements. Inasmuch as nearly all the changes from normal which we observed in our specimens, both in the glands and in the epithelium, were bilateral, we feel that they cannot be attributed to section of the fifth nerve. The only noteworthy effect might be a certain lack of mucoid degeneration of the epithelial elements on the side of section. It should be mentioned that a histologic study of the nasal mucosa of dogs which had undergone excision of the sphenopalatine ganglion and section of the superior cervical sympathetic ganglion [31] did not reveal any major results either, except for a certain lymphocytic infiltration and slight epithelial changes. No doubt these and other observations contradict the statements of Aschenbrandt [32] and Ozawa.[33]

Some sixty years ago Aschenbrandt noted a varnish-like, hyperplastic nasal mucosa a few weeks after sectioning the maxillary nerve. The blood vessels had to some extent disappeared. Extirpation of the sphenopalatine ganglion resulted only in congestion of the membrane. Extirpation of the sympathetic fibers revealed no influence at all. He attributed secretory fibers to the sphenopalatine nerves and expressed the belief that the trigeminal nerve conveys trophic fibers to the nose. To some extent, these results conform with those obtained by Ozawa. His series of rabbits were subjected to cervical sympathectomy and trigeminal denervation. He reported that intracranial avulsion was followed by desquamation

29. Karnosh, L. J., and Scherb, R. F.: Trophic Lesion in the Distribution of the Trigeminal Nerve, J. A. M. A. **115**:2144-2148 (Dec. 21) 1940.

30. Roessle, R.: Die Nasenschleimhaut, Verhandl. d. deutsch. path. Gesellsch. **19**:18, 1923.

31. Jung, L., and others: La muqueuse nasale apres excision du bloc sphénopalatin et du ganglion cervical suprême et séction du pneumogastrique. Examen histopathologique par J. F. Martin, Oto-rhino-laryng. internat. **16**:193-196, 1928.

32. Aschenbrandt: Ueber den Einfluss der Nerven auf die Secretion der Nasenschleimhaut, Monatschr. f. Ohrenh. **19**:65-76, 1885.

33. Ozawa, M.: Experimentelle Studien über den Einfluss des Funktionsausfalls von Halssympathicus und Trigeminus auf das Nasengerüst, Mitt. a. d. med. Akad. zu Kioto **10**: 758-768, 1934.

of the epithelium in the nose, formation of vacuoles and cysts and atrophy of nasal tissue, with round cell infiltration and rarefaction of the bony structure and degeneration of glands. Similar results were noted after section of the cervical sympathetic fibers. For a week the nasal mucosa was congested and reddened and showed hypersecretion, but finally widening of the nasal cavity resulted through atrophy. The epithelium degenerated and evidenced metaplasia; all layers of the mucosa were infiltrated with round cells, and crust formation was present, so that finally fibrosis of the mucosa resulted. The bone showed reduction of osteoblasts and increase of osteoclasts, infiltration of the marrow with lymphocytes and general atrophy. In his opinion, the cervical sympathetic outflow contains pain fibers. No sympathetic fibers reach the nose with the trigeminal nerve. In cases of unilateral section of the nerve the intact side also reveals changes, but only of minor degree. When both nerves are sectioned at the same time, a summation of changes resulting from their separate avulsion is produced.

Vamada's [34] observations did not support these results but tended to be in line with our own. He sectioned the nasal nerves in various combinations. In his opinion, the trophic innervation of the nose is regulated by the autonomic nervous system. Its elimination leads to atrophy of mucous glands, loosening of the propria layer and involution of cavernous tissue. The columnar epithelium is altered to squamous epithelium. The bony skeleton evidences fibrous degeneration, so that finally the turbinates atrophy. These changes are slight when the sympathetic nerve supply alone is interrupted, but when the parasympathetic nerves are also severed they are more intense. Sensory nerves have no trophic influence. The denervated nasal tissue is readily subjected to inflammatory processes, with secondary atrophy.

The nasal secretion is diminished after extirpation of the cervical sympathetic ganglion. Unilateral resection of the superior cervical ganglion in rats and subsequent study of their noses in serial sections failed to reveal any appreciable difference between the two sides.[35]

It has been established by Wertheimer and Mimoun [36] that within four months after section of the trigeminal nerve a certain hypesthesia is present, which subsides to near normal sensibility within a year. After four years sensation is supposed to be entirely normal.

Schatz [37] had the opportunity to examine 2 patients in whom the trigeminal and sphenopalatine ganglions had been resected and the cervical sympathetic and external carotid plexuses interrupted several months previously. Both patients showed disappearance of superficial sensibility for touch, pain and temperature, while deep sensibility persisted. The mucosa was unchanged, being perhaps slightly hyperplastic. Schatz stated he did not regard the sphenopalatine ganglion as a trophic nerve center. Vogel [38] observed loss of sensibility for touch, temperature and pain after extirpation of the sphenopalatine ganglion. This disturbance lasted a few months and involved the turbinates, as well as the floor of the nose, the septum and the region of the palatine nerves. No influence was noted on the injection of the nasal mucosa or on the glandular secretion, nor was the lacrimal secretion

34. Vamada, T.: Ueber die trophische Innervation des Nasengewebes, Mitt. a. d. Med. Gesellsch. zu Tokyo **47**:11, 2024, 1933.

35. Dinolt, R.: Studies on Cervical Sympathectomy in Rats, unpublished data, 1930.

36. Wertheimer, P., and Mimoun, P.: Late Results of Retrogasserian Neurotomy by the Temporal Route, J. de méd. de Lyon **19**:151-156, 1938.

37. Schatz, K. W.: Welchen Einfluss hat das Ganglion sphenopalatinum auf die Entstehung der Ozäna, Ztschr. f. Laryng., Rhin. **17**:312-315, 1929.

38. Vogel, K.: Befunde am Ganglion sphenopalatinum mit besonderer Berücksichtigung der atrophischen Rhinitiden, Ztschr. f. Hals-, Nasen- u. Ohrenh. **22**:507-553, 1929.

altered. The motor innervation of the levator palati muscle, which supposedly comes from the geniculate ganglion and the superficial petrosal nerve, was not influenced at all, nor was there any change in the secretion of sweat or in the shrinking quality of the nasal mucosa on the application of cocaine. Most likely, the nervous functions of the ganglion are taken over by the perivascular branches. This contention is not borne out by our observations that in 9 out of 10 cases, notwithstanding the interval which had elapsed since the operation, lack of normal sensibility persisted. It was suggested by Helson [39] that a return of some sensory qualities takes place when the trigeminal nerve alone is cut. Deep sensibility, localization sense and light and deep pressure sense return, while pain sensation remains lost. Temperature sensation may not be discerned correctly, heat being felt as cold and cold not being perceived at all. This observation is also borne out by the results in our case. Helson suggested that these qualities are provided for by the thoracic sympathetic fibers, or possibly in the face by the facial nerve. Especially, temperature sensation may be carried by afferent sympathetic fibers.

What influence on the nasal mucosa may one expect after section of the sensory root of the trigeminal nerve? Which fibers have been eliminated, and what effect may their interruption have on the nose? Except for the terminal nerve, the extent and influence of which on the nasal mucosa are still not sufficiently investigated, the trigeminal nerve together with the autonomic nervous system is the sole innervation of the nasal mucosa. There is no divergence of opinion concerning the macroscopic innervation. Trigeminal fibers from the first branch supply the nose through the ethmoid opening. The second branch unites macroscopically with the sphenopalatine ganglion, to which the vidian nerve also sends its fibers. Three major groups of filaments arise from the ganglion, the inner, the descending and the lateral branches. No doubt the sphenopalatine ganglion is a parasympathetic cell station, which receives its preganglionic fibers from its root, the greater superficial petrosal nerve; the majority of the fibers entering the ganglion are of the small myelinated type. A few preganglionic fibers are also carried by the sphenopalatine nerves. Further, parasympathetic fibers enter the nose with the trigeminal nerve. Postganglionic fibers run to the nasal mucosa and the lacrimal gland. Sympathetic innervation is supplied by way of the deep petrosal nerve, which passes the ganglion without interruption, and by fibers which follow the distribution of the blood vessels. Other sympathetic fibers are supplied by branches from the cavernous plexus to the gasserian ganglion, where they enter the trigeminal nerve and follow its branches (Caccialupi,[40] secretory fibers). The sensory fibers from the second branch pass to the nasal mucosa without interruption, as do the sensory filaments of the greater superficial petrosal nerve, which also contains gustatory fibers. Avulsion of the trigeminal nerve will eliminate either the entire sensory root or only one or two branches of the trigeminal nerve. With the elimination of sensory fibers a certain amount of parasympathetic fibers, and most likely the sympathetic as well, will be lost. Thus, the negative results of our observations cannot be regarded as surprising. Practically, only sensory fibers are eliminated completely, while a sufficient supply of autonomic fibers still reach the nose by way of the vidian nerve and the blood vessels to guarantee a satisfactory control of the nasal mucosa. Naturally, it is hard to judge the importance of the autonomic fibers, especially the parasympathetic, which are lost by retrogasserian avulsion. But their loss is easily made up by remaining fibers, especially as

39. Helson, H.: The Part Played by the Sympathetic System as an Afferent Mechanism in the Region of the Fifth Nerve, Brain **55**:114-121, 1932.

40. Caccialupi, P.: Il simpaticocraniano e il ganglio sphenopalatino, Riv. oto-neuro-oftal. **5**:172-188, 1928.

a great number of anastomoses exist between the sympathetic and the parasympathetic end fibers (Kümmel Jr.[40]). Perhaps the lack of these fibers is responsible for the minor degree of mucoid degeneration observed in the epithelium on the side of section. Trigeminal fibers are known to arborize within the epithelium, and part of them may be autonomic. Dryness of the nose was noted by the majority of our patients, 2 of them stating definitely that the side of operation was drier. It must remain doubtful how much importance can be attributed to this symptom, as well as to the color of the mucosa, as there is no method for its determination. Certainly, the dryness had not reached such an extent as to form heavy crusting, nor was an abnormal amount of dust noted in the noses in which removal of foreign material might be delayed by lack of moisture. Aschenbrandt observed an increase of mucoid secretion and reddening of the mucosa after section of the trigeminal nerve, and he ventured the suggestion of peripheral secretory nerve centers or of secretion by the mucosa without nerve influence, while Jung and associates [41] did not observe any changes in the appearance or secretion of the nasal mucosa of dogs during five months of observation after resection of the sphenopalatine ganglion or of the superior cervical ganglion. We believe that the pale color noted in 4 cases can be attributed to constriction of the subepithelial capillaries. Vasoconstriction is usually attributed to the sympathetic fibers, but partial elimination of the parasympathetic fibers may disturb the autonomic equilibrium sufficiently to produce a vasomotor reaction, which in 1 case even went as far as to produce shrinkage. How great a role this played in the 2 cases in which waterlogging of the mucosa was present remains open for discussion. No doubt acute vasomotor changes in the nose are liable to take place immediately after operation, partially as a result of operative shock, but our main interest must be centered in the permanent damage which may occur in consequence of section of the trigeminal nerve. Here our observations show definitely that no major influence resulted. A great number of authors have discussed the question of neurologic changes in the nose from the standpoint of ozena. The presence of ulcerations and crust formation, which older authors reported to have observed in noses with trigeminal or sphenopalatine denervation, has long supported the concept of the trophic influence of the nasal nerves. Our observations are, as far as we know, the first which have been made on a group of patients with avulsion of the trigeminal nerve on whom nasal examinations expressly intended to reveal alterations were made. The time which had elapsed since the nerve section should have given ample opportunity for the development of pathologic changes in the nose. On the contrary, among the 10 persons observed there was perhaps less evidence of active nasal disturbance or of a history of such than would be expected among a like number of persons taken at random from the same geographic region. It has been suggested that the formation of ulcerations in the nose results from a process similar to that of neuroparalytic keratitis. The number of sympathetic nerves sectioned together with the fifth nerve is thought to be of importance. Ulceration was also observed more frequently in regions in which there was an excellent blood supply. No doubt nasal branches of the trigeminal nerve do not carry a sufficient number of sympathetic nerve twigs to be of great importance, and in spite of the loss of pain, and to some extent of touch sensation, the patient is able to avoid lesions to the nasal mucosa. The anesthesia observed in cases of ozena is certainly a secondary affair, due to degeneration of the nerve endings in the dystrophic membrane and not to primary dysfunction of the trigeminal nerve.

41. Jung, L.; Tagand, R., and Chavanne, F.: Sur la role trophique des nerves sécrétoire de la muqueuse nasale, Compt. rend. Soc. de biol. **99**:515-516, 1928.

No inference should be drawn from this statement as to the role of the autonomic nervous system in trophic control of the nose. The limits of its effectiveness have not been definitely established, nor have all of its connections been determined. Most authors neglect the possibility of another autonomic nerve pathway to the nose, namely, the terminal nerve. The rich supply of autonomic fibers in this nerve and its interlacing connections with trigeminal branches suggest relays of impulses, a possibility which has been neglected in most experimental denervations. Therefore, our investigation, conducted on a small scale, shows that sensory denervation of the nasal mucosa produced by the retrogasserian avulsion of the sensory branch of the trigeminal nerve does not have any major influence on an eventual pathologic condition of the nose nor does it result in any permanent changes in the mucosa.

SUMMARY

The innervation of the nasal mucosa and some aspects of the relations of the sphenopalatine ganglion are discussed.

The nasal history, the observations on the nose and the results of biopsy of tissue taken from the turbinates of 10 patients in whom the sensory root of the trigeminal nerve had been sectioned five months to thirty-nine years previous to this investigation are reported.

Neither the histories nor the observations permit any special conclusions to be drawn. Anesthesia of the affected side was noted in 9 patients and hypesthesia in 1 patient. No major pathologic change was noted. Histologic study failed to reveal any differences between the two sides of the nose, except for a possible decrease of mucoid degeneration in the nasal epithelium on the side of operation. Neither the vessels nor the glands evidenced changes which could be attributed to the lack of trigeminal fibers, nor was there a perceptible change in nasal secretion, ulceration or crust formation.

The possibility that the terminal nerve as a part of the autonomic nervous system exerts a greater influence than has been generally considered is suggested.

It has been established that trigeminal denervation is not followed by either temporary or permanent damage to the nasal mucosa, nor does it predispose the person to a pathologic condition of the nose.

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ACUTE MASTOIDITIS MASKED BY TREATMENT WITH SULFONAMIDE COMPOUNDS

CLINICOPATHOLOGIC CONSIDERATIONS

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Much has been written in the past few years attesting to the general use of sulfanilamide and its derivatives in acute pathologic conditions of the ear. As conservative therapeutic agents in the treatment of purulent otitis media and mastoiditis the status of these compounds is still debatable. Fenton [1] asserted that sulfanilamide was an invaluable agent and expressed the belief that operation on many "red hot" ears has been avoided by its use. Babcock [2] stated that its effect in the treatment of acute otitis media is overrated. Bowers [3] expressed the opinion that it increases the chance of prompt resolution by 50 per cent and decreases the chance of operation by 50 per cent. Scal [4] saw no value in the drug if bone destruction exists, and Maybaum and associates [5] reserved it for the treatment of serious complications.

There is common agreement among Fenton,[1] Maybaum and associates,[5] Scal,[4] Converse,[6] Bowers,[3] Lillie,[7] Babcock,[2] Kopetzky [8] and Maxwell [9] that a change in the familiar signs and symptoms of mastoiditis can occur after the administration of a sulfonamide compound. Quiescence in the clinical signs gives the impression that the infectious process is resolving. This masking causes a dangerous false sense of security. Since this change is caused by chemotherapy, Kopetzky,[8] Galloway,[10] Bowers,[3] Lillie [7] and Williams and associates [11] have suggested the adoption of a conception of what constitutes operative mastoiditis in a patient

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1. Fenton, R. A.: Sulfanilamide in Otolaryngology: Some Personal Experiences, Ann. Otol., Rhin. & Laryng. **48**:17 (March) 1939.

2. Babcock, J. W.: Role of Sulfanilamide in the Treatment of Acute Otitis Media, Arch. Otolaryng. **32**:246 (Aug.) 1940.

3. Bowers, W. C.: Observations on Seven Hundred and Ninety-Three Cases of Acute Purulent Otitis Media, J. A. M. A. **115**:178 (July 20) 1940.

4. Scal, J. C.: Evaluation of Sulfanilamide Therapy in Acute Otitis Media and Mastoiditis, New York State J. Med. **39**:1790 (Sept. 15) 1939.

5. Maybaum, J. L.; Snyder, E. R., and Coleman, L. L.: Experiences with Sulfanilamide Therapy for Otogenous Infections, Arch. Otolaryng. **30**:557 (Oct.) 1939; The Value of Sulfanilamide in Otogenous Infections, J. A. M. A. **112**:2589 (June 24) 1939.

6. Converse, J. M.: Recurrence of Otitic Infections Due to the Beta-Hemolytic Streptococcus Following Inadequate Sulfanilamide Therapy, J. A. M. A. **113**:1383 (Oct. 7) 1939.

7. Lillie, H. I.: Treatment of Acute and Chronic Suppurative Otitis Media, J. A. M. A. **115**:506 (Aug. 17) 1940.

8. Kopetzky, S. J.: Purulent Otitis Media, Mastoiditis, Sinus Thrombosis and Suppurations of the Petrous Pyramid, Arch. Otolaryng. **32**:962 (Nov.) 1940.

9. Maxwell, J. H.: The Sulfonamide Drugs in the Treatment of Acute Suppurations of the Middle Ear and Mastoid, Surg., Gynec. & Obst. **74**:573 (Feb.) 1942.

10. Galloway, T. C.: Chemotherapy, Arch. Otolaryng. **30**:901 (Dec.) 1939.

11. Williams, H. L.; Brown, A. E.; Herrell, W. E., and Ralph, R. D.: Sulfonamide Therapy for Acute Otitis Media and Mastoiditis, Ann. Otol., Rhin. & Laryng. **50**:336 (June) 1941.

receiving one of the sulfonamide compounds. This can be achieved if the modified clinical picture is considered an entity and acknowledged as such, rather than as a transformation of established pathologic impressions.

PATHOLOGIC PROCESS

Acute infections of the middle ear and mastoid exhibit variable bacteriologic features. The common pathogen is the hemolytic streptococcus. Next in frequency are the pneumococci, especially type III, the nonhemolytic streptococcus and Staphylococcus aureus. The incidence of the influenza bacillus and of mixed infection is small.

The first reaction to infection of the mucous membrane lining the tympanum and the cells of the mastoid process is manifest edema. A serous effusion then fills the bony cavities, and leukocytic infiltration of the vascular mucoperiosteum follows. Migrating white cells change the fluid from serous to purulent. The rapidity with which this expands and the amount of fluid present depend on the virulence of the invading organism. Pressure by this purulent material induces necrosis and destruction of the mucous membrane, the mucoperiosteum and the bony trabeculae of the mastoid cells. The destroyed epithelium is replaced by granulation tissue, infected at its site of origin. Retrograde thrombophlebitis of the small veins in the mastoid occurs early or late in the course of the infection, and additional tissue dies from insufficient blood supply. The periodicity of infected clot formation is a factor in producing a hemorrhagic or a coalescent type of mastoiditis.

The advance of this pathologic process, and in favorable cases the healing, is uniform throughout the mastoid cells and the tympanic cavity. Resolution is accompanied by the organization of granulation tissue and regrowth of bone. In young children further pneumatization is inhibited. The vascularity of the part always suffers, and subsequent infections are apt to be more severe.

Ballenger and associates [12] stated that nitrogen, essential to the metabolism of most pathogens, is obtained from peptone-like bodies present in pus and tissue undergoing necrosis. By their destructive action the causative bacteria in cases of otitis media and mastoiditis produce an environment suitable to sustain their virulence and at the same time hinder the bacteriostatic action of the sulfonamide compound. This action is further impaired by a lowering of the already meager supply of blood and oxygen to the infected area. The quick response which characterizes the action of the new chemotherapeutic agents on diffuse nonsuppurative infections may not be obtained in an active inflammatory lesion of bone.

In acute mastoiditis treated with a sulfonamide compound the infection is often only partially controlled and continues to smolder. Galloway [10] and Babcock [2] observed a mottled pathologic process. Areas of softening and abscess formation, from which positive cultures were obtained, were surrounded by normal bone. Granulation tissue showing evidence of organization and new bone formation in cells adjacent to coalescent portions were observed. The bony septums containing pus-filled cells may be devoid of mucosa or granulations and resemble nonsequestrated, dead bone.[2] This "spotty" [10] necrosis has an affinity for the dural and sinal plates, with perisinal or epidural abscesses as natural sequelae.[3]

12. Ballenger, E. G.; Elder, O. F.; McDonald, H. P., and Coleman, R. C., Jr.: Failures in the Treatment of Urinary Tract Infections with Sulfanilamide, J. A. M. A. **112**:1569 (April 22) 1939.

CLINICAL CHARACTERISTICS

The signs and symptoms of acute mastoiditis under the conditions of this therapy are subject to minor variations, dependent on the micro-organism present or the amount of the sulfonamide compound administered. The speed of the infectious process is retarded, and a clinical picture almost identical with mastoiditis due to the type III pneumococcus is produced. To evaluate properly this atypical disease, all diagnostic aids at one's command should be employed.

The amount of aural discharge is variable. It may be scant or moderate, with profuse drainage likely to occur when the therapeutic agent is discontinued. Sterile otorrhea is frequently encountered.

Examination of the tympanic membrane reveals persistent thickness, with the short process visible at times. Sagging of the posterosuperior wall of the canal or prolapse of the drum membrane is seldom detected. Severe reduction in hearing acuity, of the conductive type, is present.

Pain and tenderness over the mastoid are absent.

The temperature is erratic. Early administration of the drug causes a prompt drop to normal, followed by irregular elevation. Pyrexia is a frequent manifestation of an allergic reaction to the sulfonamide compounds, and during the administration of the drug the bearing of the temperature on the course of the disease suffers accordingly. The characteristic variations in temperature denoting invasion of the meninges or thrombophlebitis of the lateral sinus are absent.

Law and Taylor, in personal communications to Scal,[4] pointed out the change in density to roentgen rays that takes place in the mastoid with the administration of these compounds. The bony septums appear thinner and the cell contents more translucent. This decreased opacity makes it difficult to evaluate properly a single picture; therefore stereoscopic films are essential. The authors suggested that the roentgenologist should be notified that the drug has been used.

Infection always alters the blood picture. In cases in which acute mastoiditis is treated with a sulfonamide compound this change is further influenced by the agent employed. All sulfonamide compounds tend to decrease the white blood cell count. Fenton[1] stated that a concomitant lowering of the red cells may signify beginning hemolytic anemia. Leukocytosis, with a "shift to the left," is neither constant nor characteristic in this modified clinical picture. Discontinuation of the drug results in a rapid elevation of the white cell count.

Positive cultures of the blood and spinal fluid are seldom reported. According to Converse,[6] these sterile cultures result from the planting of material in culture mediums incapable of diluting the concentration of the drug beyond its minimum bacteriostatic point. This bacteriostatic effect can be neutralized by the addition of 5 mg. of paraaminobenzoic acid to each hundred cubic centimeters of medium.

Systemic symptoms, such as chills or chilly sensations, cerebral disorientation and headache, formerly considered in the light of the inflammatory lesion of the mastoid bone must now be regarded as possible toxic phenomena of the therapeutic agent employed.

Animals fail to form protective antibodies to streptococcic infections if treated with sulfanilamide.[6] The frequent relapses of symptoms in cases of mastoiditis treated with the sulfonamide compounds may be explained by this fact. The patient does not acquire a natural immunity, and the organisms regain their former virulence when the concentration of the drug is lowered.

The alteration in the pathologic picture caused by the sulfonamide compounds is perplexing. The amount of the destruction observed at operation is more extensive than is implied clinically. Prolonged conservative therapy may increase the potentiality of danger.

Clearcut symptoms indicating the necessity for surgical intervention are absent. Operation is deemed advisable with fewer symptoms and signs than are present in cases of surgical mastoiditis unaltered by the administration of a sulfonamide compound.[8]

SUMMARY

1. The clinical picture in which the symptoms of acute mastoiditis are masked by treatment with a sulfonamide compound is discussed.

2. The pathologic and clinical features of this modification are compared with surgical mastoiditis unaltered by chemotherapy.

Dr. Clarence H. Smith made suggestions in the preparation of this article.

2940 Grand Concourse.

THE DEVIATED SEPTUM

PHYSICOPHYSIOLOGIC ASPECT

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Nasal stenosis due to deviation of the septum has received scant attention in the general or specialized medical literature in recent years. Although persons with this abnormality are many, they rarely seek medical advice for it. They learn to disregard it or simply "put up" with it. Not until secondary nasal or systemic complications develop is the physician consulted. The consequences of mechanical nasal obstruction are manifold and not infrequently of serious nature. This, however, is not appreciated by members of the medical profession in general because opinion rather than *de facto* knowledge is the basis for judgment. Rhinologists consider a submucous resection of the nasal septum a routine measure, for which they have developed their own subjective judgment regarding its indication, as well as its technical execution. Such an attitude tends to depreciate the value of this most beneficial operation and brings confusion into the mind of the student of rhinology. Moreover, it has created a general antagonism among members of the medical profession outside of rhinology and has led to general condemnation of this and other nasal surgery. Though it is true that a submucous resection of the septum, when wrongly indicated or performed, may result not only in no improvement but in ill immediate and late consequences of permanent nature, it is likewise true that a properly performed operation in a case in which there is thoughtful indication brings immeasurable relief to a patient locally as well as constitutionally. The result is not only of immediate but of inestimable value as a prophylactic measure for nasal and sinal disturbances and their far-reaching constitutional consequences. The discussion of the physicophysiologic aspect of mechanical nasal obstruction and its etiologic relationship to local and distant organic pathologic conditions is the object of this presentation.

Generally speaking, it can be said that the indication for a submucous resection should be dependent not on the deviation of the septum from the median sagittal plane but on the degree of obstruction this effects. These two conditions do not necessarily coincide. A straight but thickened septum in an otherwise narrow bony nasal frame can be more obstructive than a severely bent and deformed septum in an otherwise wide nasal cavity. Correction of the first defect will be most beneficial, while correction of the second may be harmful in that it will unduly increase the width of the nasal cavity. This, as is explained later, not only causes the local discomfort of atrophic rhinitis but produces detrimental changes in the larynx and the lower respiratory tract of a permanent nature. Therefore an appraisal must be made of the degree of patency of the nasal passages, and not of their configuration only.

A simple device for this purpose was described by Zwaardemaker [1] in 1893 and has since been modified [2] and adapted for quantitative measurement. Basically the test consists of the following steps: A mirror or a polished metal plate is

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1. Zwaardemaker, H., and Reuter, C.: Athembeschlag als Hülfsmittel zur Diagnose der nasalen Stenose, Arch. f. Laryng. u. Rhin. **1**:176, 1893.

2. Silcox, L. E.: Rhinitis: A Nasographic Study, Arch. Otolaryng. **34**:33 (July) 1941.

cooled by immersion in cold water. It is then dried and placed horizontally under the nose and against the upper lip. The patient is asked to exhale through the nose. This produces a pattern of frosting on the mirror. The difference in the size of the pattern for each nostril represents their difference in patency, or, more correctly, the amount of resistance encountered by the exhaled air. More accurate information, which also includes the recording of the degree of patency on inhalation, can be obtained by placing a tube inside the nose and connecting this with a manometer.[3]

Nasal respiration has two phases: inspiration and expiration. The first produces a negative and the second a positive pressure in the nasal cavity. The effect of that rhythmic change is twofold: First, it helps maintain normal physiologic conditions in the sinus cavities by the promotion of aeration and drainage. The vacuum in the main nasal chamber forces air from the sinus cavities, and the expiratory stream of air, when passing the ostiums of the sinuses, siphons secretion from the sinuses on the same principle on which the fluid empties from the barrel of an atomizer. Second, it controls to an appreciable extent the patency of the nasal meatuses by its effect on the turgescence of the turbinates. The latter is conditioned by the degree of filling of the pseudocavernous spaces, which are particularly numerous in the inferior turbinate and the vascular network. The rhythmic respiratory change affects the state of active or passive congestion of these blood vessels in a way which is similar to the influence of the respiratory cycles in the thorax on the action of the heart. This fact has not received the appreciation it deserves. By its regulatory action on the volume and pressure of the arterial blood column, the respiratory cycle in the nasal cavity further influences the activity of the nasal glands. These glands, as will be recalled, produce the mucous blanket which protectively covers the mucous membrane and provides the surface moisture, which is all-important for the maintenance of normal ciliary motion.

It was explained in a previous publication,[4] which deals with the rationale for surgical narrowing of the nasal air passages in cases of ozena, how an abnormally wide nasal frame is responsible for bringing about and furthering the atrophy, and how surgical restoration of the normal width stops the atrophic process. An abnormally wide nasal passage, because of the diminished resistance, subdues the amplitude of the excursions of the inspiratory negative and the positive expiratory pressure. This, in turn, decreases the turgescence of the turbinates by diminishing the blood supply to the mucous membrane and its adnexa, the glands in particular. The large volume of air which passes over the surface of the mucous membrane evaporates the already reduced amount of secretion, which causes stagnation of ciliary motion, with all its harmful consequences. In cases of extreme atrophy the resistance to the inhaled air is as low as that of the oral cavity, where the rhythmic pressure changes are negligible. The nose then loses its efficiency as air conditioner for the lungs; i. e., it does not warm, humidify and filter the inhaled air of solid particles. The local effects are impairment of aeration and drainage of the sinuses, and retardation of normal pneumatization of the facial skeletion if the condition is present in early life, before the sinuses are fully developed.

If the volume of the nasal cavities is reduced, as in the case of an obstructing septal deviation, then the inhaled air encounters greater resistance. The amplitudes of the respiratory negative and positive pressures will be greatly increased if a normal

3. Sternstein, H. J., and Schur, M. O.: Quantitative Study of Nasal Obstruction: A New Method, Arch. Otolaryng. **23**:745 (April) 1936.

4. Wachsberger, A.: A New Technic in the Surgical Approach of Ozena, Arch. Otolaryng. **19**:370 (March) 1934.

volume of air—about 12 liters per minute—is to be supplied to the lungs. This, unless relieved by partial mouth breathing, causes an undue strain on the tonus of the blood vessels, which in time respond with paretic relaxation. The result is chronic engorgement of the turbinates, which adds to the already present stenosis due to the septal block. A vicious cycle is started. The truth of this clinical observation was recently verified by Sternstein [5] in experimental investigations. He made manometric readings of the resistance to the respiratory air current in a normal nose. After forcing the air through the meatuses under artificially increased pressure for a certain period, he found at the end of that period an increase of resistance to normal respiration and a lowered p_H of the nasal secretion. The mucosa, especially of the turbinates, showed considerable engorgement. If this swelling affects the membranous lining of the sinus ostiums, interference with maintenance of their normal physiologic activity may result. In conformity with this assumption is my [6] observation that actual hypertrophy, with formation of polyps, resulted from the surgical reduction of the nasal volume in cases of ozena, in which the mucous membrane was in a state of extreme atrophy previously.

Nature has supplied a most sensitive and complicated mechanism for the purpose of regulation of the patency of the nasal passages. This mechanism is reflectorily conditioned by exogenous factors, such as temperature, water content, contamination with solid particles or gases of the outside air, atmospheric pressure and wind velocity, and by endogenous factors, such as exercise or rest, digestion and emotional reflexes. In fear the nostrils dilate and the turbinates pale and shrink, and they become engorged during crying. Secretion from the glands increases, and its consistency decreases. A normal nose is in a constant state of alert and is subject to continuous changes if it is to serve well not only as the olfactory organ but, what is more important in human beings, as the air conditioner for the organism.

With full realization and knowledge of the normal and pathologic physiology, the rhinologist must determine the specific indication for a submucous resection in the given case. He should not and cannot judge the normal state of patency when examining a patient during an acute attack of inflammation or vasomotor alteration for which he may be consulted at the time. The indication for correction should preferably be deferred to a subsequent examination, when the acute condition has subsided and the patient again experiences his normal nasal status.

It is similarly fallacious to judge nasal patency during an acute or a chronic state of swelling of the turbinate bodies, for a minor deviation of the septum may seem obstructive if the conchae, especially the inferior, are in contact with the septum during such a disorder. Inflammatory and allergic enlargement of the turbinate bodies, however, must be differentiated from true hyperplasia, i. e., a numerical and voluminous increase of the cell elements of the mucous membrane and the turbinate bones. Such hyperplasia is frequently observed on the inferior turbinates on the concave side of the septal deviation and has been termed compensatory. Pseudohypertrophy must also be ruled out. This term was applied by Sternstein to describe a turbinate which projects into the nasal cavity and simulates hypertrophy of the mucous membrane. Actually, it is the turbinate bone which, instead of curving downward to the nasal floor from its insertion into the lateral nasal wall, projects in a more obtuse angle across the inferior meatus toward the septum.

5. Sternstein, H. J.: Quantitative Effects of Physical and Chemical Agents on the Erectile Tissue Response in the Nose, Arch. Otolaryng. **34**:523 (Sept.) 1941.

6. Wachsberger, A.: Surgical Correction of Atrophy in Ozena, S. Clin. North America **18**:487 (April) 1938.

Simple lateral infraction along the insertion corrects the obstruction and verifies the diagnosis of pseudohypertrophy. This procedure will not correct stenosis due to a truly hypertrophic turbinate.

The usual practice of shrinking the turbinates with cocaine or epinephrine and then judging the patency of the nasal air passages is not an entirely reliable way of determining the indication for surgical correction of a septal deviation, because, as has already been seen, an obstructive septum may cause and support chronic turgescence of the turbinates, which will temporarily disappear after the application of one of these drugs. A submucous resection, on the other hand, will bring about permanent adjustment of the tonus and vascularization of the turbinates.

Tonus as applied to the inferior turbinate is a term not generally used; yet it seems most descriptive. It signifies the balanced degree of contractility of the fleshy parts of the turbinate, especially the irregularly arranged smooth muscle and elastic connective tissue fibers which are present in the stroma surrounding the pseudocavernous blood vessels. They help control the degree of filling of these spaces and therewith the size of the turbinate to a large extent. This will be appreciated if it is remembered that the pseudocavernous bodies are fed by veins and have to be emptied passively, while the true cavernous bodies in the sex organs are fed by arteries. There, a diminished arterial pressure will cause these blood cavities to empty. In the nose a sensitive reflex action is required of the muscle and elastic fibers in order that the demands of the changing exogenous and endogenous factors may be complied with. A continued strain on these structures, as conditioned by sustained inflammation, allergic edema or mechanical obstruction, paralyzes their contractility. The turbinate then appears as a sagging, flaccid body and presents the color characteristic of passive congestion. Application of epinephrine does not readily reduce its size. It has lost its tonus.

The obstructiveness of the turbinates must therefore be judged per se, as well as in relation to the deviation or undue thickening of the septum, when one is considering to what extent the septum alone is responsible for the stenosis and whether the size of the turbinates is etiologically conditioned by the septum. If the turbinates are truly hypertrophic as a whole or if they present circumscribed redundancies, as is often the case on the posterior tip, then their reduction either surgically or with cautery will suffice. Conservatism must be practiced in this accomplishment. Only a little at the time must be done and the effect of subsequent scar contraction observed before a greater reduction is undertaken. It is easier to widen a narrow lumen than to decrease it.

Septal deformities occur in the greatest variety. The most bizarre shapes and contours are observed, even without the history of fracture, which not infrequently complicates the condition. It can be said that the most obvious and most frequent departures from the sagittal midline plane interfere least with breathing. Reference is made to the ridges and spurs at the juncture of the quadrangular septal cartilage with the vomer. These are usually situated near the nasal floor opposite the anterior third of the inferior turbinate and are easily seen without previous shrinking of the turbinate. They are of lesser significance because they interfere little with the normal course which the respiratory stream takes, and therefore do not interfere with olfaction and aeration and drainage of the sinuses. If they are extremely large and protuberant or if they extend far backward, they may disturb normal nasal function. The vomer increases in height posteriorly. Projections in this region may obstruct the middle meatus. Their significance is controversial and cannot be ascribed to their obstructiveness. They are, however, held responsible for reflectory symptoms, such as vasomotor disturbances

or neuralgia of the sphenopalatine ganglion,[7] when they extend into the inferior turbinate or reach the lateral nasal wall posterior to the posterior tip of the middle turbinate, where the sphenopalatine ganglion is situated directly under the mucous membrane. Occasionally these projections interfere with the ventilation of the eustachian tube.

Deviations in the area of the middle meatus are of much greater significance. They are not seen on superficial examination and can be visualized fully only after previous spraying with a decongestant. It will be remembered that the inhaled air normally ascends from the vestibule to the anterior tip of the middle turbinate, passes through the middle and superior meatuses and then downward through the nasopharynx into the lower respiratory tract. In a case in question the air current is deviated through the inferior meatus. Since all sinus ostiums are situated in the middle and superior meatuses, the effect of the respiratory cycle on aeration and drainage of the sinus cavities is eliminated. Furthermore, the sinus ostiums are crowded by the middle turbinate, which is pressed laterally and over them by the septal deviation. This predisposes to infection of the sinuses. Olfaction, unless compensated by the other side, is also impaired because of the obliteration of the olfactory fissure.

Another form of deviation, which deserves attention but is frequently overlooked on rhinoscopic examination, is a bend in the most anterior part of the septal cartilage. This is often termed subluxation of the septum, because it frequently is combined with a displacement of the inferior rounded edge of that cartilage from the groove of the vomer, where it normally rides like a railroad wheel on the track. A nasal speculum, when inserted, covers the deformity. Examination of the nostril without an instrument, however, makes the site of the obstruction obvious. If the patient is asked to inhale sharply through the nose, it will be noted that whatever chink may be present on quiet respiration will be obliterated by the aspiration of the ala nasi, which is brought about by the vacuum in the nasal cavity on inhalation through the other nostril. This vacuum is present at all times during inhalation, though to a lesser degree than during this experiment. On exhalation the amplitude of the positive pressure is also increased because of the increased resistance encountered at the nostril. The deleterious effect of the continuous increases in the amplitudes has already been explained.

Rhinoscopic examination must include inspection of the area of the tuberculum septi. This is situated most anteriorly, at the junction of the septal cartilage and the nasal bones, where the latter join with the upper lateral cartilages. The projection of this site on the external surface of the nose corresponds to the midsection between the bridge and the tip. The tuberculum is represented by a thickening of the mucous membrane, which histologically is composed of rudimentary cavernous bodies. It is extremely sensitive. The nasociliary nerve passes in its immediate vicinity from the inside of the nose to the skin of the nasal tip as the nervus nasalis externus. Sneezing and tearing can be elicited in some persons by pressure of the finger on the area from the outside. If owing to excessive width or deviation of the underlying cartilage, the tuberculum is in continuous apposition with the lateral nasal wall, the contact may be the source of a chronic reflectory vasomotor reaction of the nasal tissues. If this contact is released by the removal of the cartilage and the tuberculum itself is destroyed by scarification, cessation of the vasomotor symptoms can be expected; this was observed in many cases.

7. Eagle, W. W.: Sphenopalatine Ganglion Neuralgia, Arch. Otolaryng. **35**:66 (Jan.) 1942.

Aberrations of normal nasal respiration exert a harmful effect not only on the nasal structures and sinuses but on the entire respiratory tract and, through the latter, on the entire organism.

The cavities of the middle ear are pneumatic offshoots from the nasal chambers, just as the sinuses are considered to be. The difference lies, first, in a long tubular entrance, which is in part controlled by muscular contraction, and, second, in a nonrigid fundus, the drum membrane. The dependence of regulated air pressure in the middle ear on normal ventilation of the nose is sufficiently appreciated. Aviation and underground construction have brought this problem to the fore of late with the observation of what is called the blocked ear and aero-otitis. Correction of abnormalities in nasal respiration is of prime concern in the prophylaxis and therapy of these conditions. Attention must be paid to the fact that it is not only the mechanical obstruction of the nose which influences the middle ear but the chronic catarrhal alteration of the mucous membrane which establishes itself as a result of the former and invades the mucosa of the eustachian tube and the cavity of the middle ear. Conduction deafness and tinnitus may result.

Normal hygiene of the nasopharynx is also dependent on nasal respiration. Acute colds most frequently start with inflammation in the vault of the nasopharynx. If one side of the nose is obstructed, then all the air supply for the lungs must pass through the other, with the result that the air is not sufficiently conditioned and impinges thus on the mucosa of the nasopharynx. It does so with great force, since the same amount of air which normally flows through both sides is coerced to flow through half the size of the tube normally available. Although this causes a chronic state of mild irritation—chronic postnasal drip—during normal atmospheric conditions, it suffices to produce acute inflammation in cold and dry winter weather or in the dry air of steam-heated apartments. In the case of a deviation of the septum in the area of the middle meatuses, the same condition will result, because the air current must take a much shorter and more direct course through the inferior meatus, instead of the circuitous normal route. The etiologic relationship between frequent attacks of coryza and stenosis of the nasal passages is thus established.

Oral-pharyngeal disorders are rarely directly traceable to nasal stenosis only. The pharynx is lined with stratified squamous epithelium, which is much less vulnerable than the ciliated respiratory epithelium. Mouth breathing does dry the pharyngeal mucosa, especially during sleep, when the salivary glands are less active. The results of abnormally wide nasal passages, such as are present with atrophic rhinitis, are more often observed in the pharynx. The typical dry, varnished appearance of the posterior pharyngeal wall is well known.

The larynx and trachea, however, are greatly influenced by impaired nasal respiration. They are, to the greatest extent, lined with respiratory epithelium. The vocal cords are covered with stratified squamous epithelium. They do not contain any glands themselves, but require an adequate supply of moisture for voice production and the elimination of unwarranted cough reflexes. For the supply of surface moisture they depend on the surrounding structures, especially the secretory glands in Morgagni's ventricle. Chronically blocked or unduly patent nasal passages cause a dry, chronic inflammatory irritation, with hawking, cough and hoarseness. In ozena the crusts in the nose, which form as a result of increased ventilation and evaporation, usually extend to the larynx and the upper part of the trachea. The nasal and sinal cavities are known to function as resonators for the voice. While little effort is required on the part of the larynx for the voice to be "carried" in a person with normally patent nasal passages, this effort must be multiplied greatly by a speaker with a clogged nose. Paresis

of the laryngeal muscles, evidenced by easy tiring of the voice, results. In professional singers or speakers who continuously make a forceful effort in order to overcome such weakness, callouses develop on the vocal cords in the form of singers' nodules or a more extensive pachyderma. They disappear spontaneously if the septal deviation is corrected and vocal hygienic measures are instituted.

The respiratory excursions of the thorax are reflectorily conditioned by the nose. Clinical proof for the existence of this reflex is frequently observed by the rhinologist. Adults with a marked deviation of the septum, if the condition has existed before puberty, almost invariably have the typical "sunken-in" thorax, with a short anterior-posterior diameter. Also, shortness of breath on mild exertion, and even asthmatic attacks, have been alleviated in cases of nasal stenosis with the removal of the mechanical obstruction. Because of its impeding effect on chest expansion, especially during growth, nasal obstruction should be corrected if and when it is observed in early life, instead of the operation being postponed until the age of 18, as is usually the practice.

Indirect and harmful effects of nasal stenosis on distant organs will be appreciated when the interdependence of thoracic movement and heart action is remembered. Discussion of these aspects is beyond the scope of this paper.

One more aspect of impaired nasal respiration should be stressed because it is rarely mentioned in textbooks, that is, its effect on coordinated cerebration. That aprosexia nasalis, a lack of attention and mental concentration in children, is due to nasal obstruction from hypertrophied adenoids has long been appreciated. In adults, however, this effect has been underestimated. Every one who has suffered from a stuffy nose during a head cold has experienced a certain dulness of the sensorium and hyperirritability. This derangement is not due to the intoxication from the infection as much as to blocked nasal passages and is quickly, though temporarily, relieved with the application of a decongestant. The same state of mind, though to a lesser degree, is observed in adults with obstructing septal deviations. The appreciation for the relief afforded by surgical correction is great in intellectual patients.

A submucous resection of the septum is a minor operation. It is performed with local anesthesia and when the anesthetic is properly administered is entirely painless and without postoperative pain. Discomfort, if any, is experienced during the first twenty-four hours, when both nasal cavities are packed with gauze so as to attain apposition of the mucoperichondrium of both flaps. This discomfort is relieved by the insertion of thin rubber breathing tubes beside the packing. The patient need not be incapacitated for more than three to five days. The postoperative period is considerably shortened if the operation is performed when the patient shows the least possible signs of irritation. It must never be performed in the presence of an acute inflammation.

CONCLUSION

Local and systemic consequences of nasal obstruction from septal deviations are largely due to the physical factors involved. These have been elucidated.

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EXPERIMENTS ON CONDUCTION OF SOUND THROUGH THE CAVITY OF THE MIDDLE EAR

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The performance of the ear as a receiver of acoustic energy is most commonly expressed by determination of the threshold. Without question, the electrical sound producer represents a great improvement over older devices for hearing tests. However, it becomes increasingly evident that the threshold of hearing alone does not completely represent the state of auditory function. For a number of years, especially since the work of Fowler, more attention has been paid to the function of the ear with regard to overthreshold stimuli.

It has been suggested that an acoustic receiver capable of conducting such a multitude and variety of intensities is most likely not uniform in its mechanism. This assumption was made both as to frequency and as to intensity. There is experimental evidence that the mode of vibration for very high intensities close to the threshold of feeling is different from the mode of vibration of intensities near the threshold of hearing. For example, von Békésy [1] reported that the stapes changes its axis when the intensity becomes great. Instead of pivoting at its posterior pole, the stapes rocks along the long axis of the foot plate. This change of vibration is said to reduce the resulting movements of the fluid in the inner ear, and serves therefore as a protective mechanism.

In this paper the question of variable sound conduction is studied experimentally for a limited number of middle range frequencies. Experimental evidence is presented which shows that the ear does not maintain a uniform conducting mechanism throughout the scale of intensities. The emphasis is based on experimental demonstration that ossicular sound conduction can shift to paraossicular sound conduction at different levels of intensity. Paraossicular conduction is defined as conveyance of sound energy to the inner ear by any route other than the ossicular chain. The term aerocochlear conduction was coined by Alexander to describe the conduction of sound through the air of the middle ear cavity to the round window.

The problem was studied both in the animal and in man. There were also experiments carried out on fresh human temporal bones.

As described in a previous paper, it is possible to measure the acoustic stimulation of the cochlea in animals by observing the reflex contractions of the tensor tympani muscle. In our experiments on aerocochlear sound conduction the middle ear of the animal was exposed in a similar way, as shown in figure 1. The opening of the bulla ossea can be closed by a glass cover. The muscles of the middle ear can be observed through this window. Readings can be taken with the bulla open

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1. von Békésy, G.: Akust. Ztschr. **25**:413, 1936.

or closed. In the former condition sound can enter the ear through the outer acoustic canal and also through the bulla ossea directly. In the latter sound can reach the cochlea only through the outer ear.

DEMONSTRATION OF AEROCOCHLEAR SOUND CONDUCTION IN THE NORMAL ANIMAL

Figure 2 *I* shows the threshold of the tensor tympani reflex with the bulla closed. The curve represents the normal conditions of sound conduction, acoustic energy entering the ear through the pinna and the outer auditory meatus. If the lid on the bulla is removed and the threshold read again no change is observed. For threshold intensities there is no difference if sound energy is permitted to reach the cochlea through the air of the middle ear. This observation confirms the former statement of Lorente de Nó that the opening of the bulla has no influence on the threshold of the tensor tympani contraction.

Fig. 1.—Photograph of the middle ear as exposed in the animal experiment. The incision was made parallel to and about 2 cm. lateral to the medial line of the neck. The bulla ossea was exposed and opened. All important parts of the middle ear are accessible through this opening.

Sound waves can reach the cochlea directly through this hole in the bulla ossea. If the opening is closed with glass the conduction of sound waves is made impossible; the tensor tympani reflex, however, used as an indicator of cochlear function, can still be observed.

Experiments with overthreshold intensities, however, gave a completely different result. For the measurement of overthreshold contractions of the tensor tympani muscle the following optical recording device was used. A small steel hook was connected with the belly of the muscle. Through a silk thread the hook in the muscle was connected with a little wheel, on the axis of which a mirror was mounted. If the tensor tympani muscle contracted it pulled on the hook and thus on the wheel. The turning of the axis was demonstrated by a light beam reflected from the mirror. By this method it was possible to record the strength of the muscle contraction for each tone intensity. Figure 2 *II* shows the muscle contraction as a function of tone intensity. The influence of the aerocochlear pathway is demonstrated in the curve.

Another method for overthreshold tests consisted in comparison of the reflex contractions for ossicular and for aerocochlear conduction. First, the threshold of the tensor tympani reflex was determined. Then a 10 decibel overthreshold tone was sounded while the bulla window was open. The strength of the contraction was determined with this stimulus. The intensity of the tone which produced the same contraction while the bulla was closed was measured. For various intensities and at different frequencies the measurements could be recorded in the way seen in figure 3 *I*. The oblique lines demonstrate that less tone intensity is required to produce a given reflex contraction if the sound is permitted to enter the cochlea through the outer and the middle ear simultaneously.

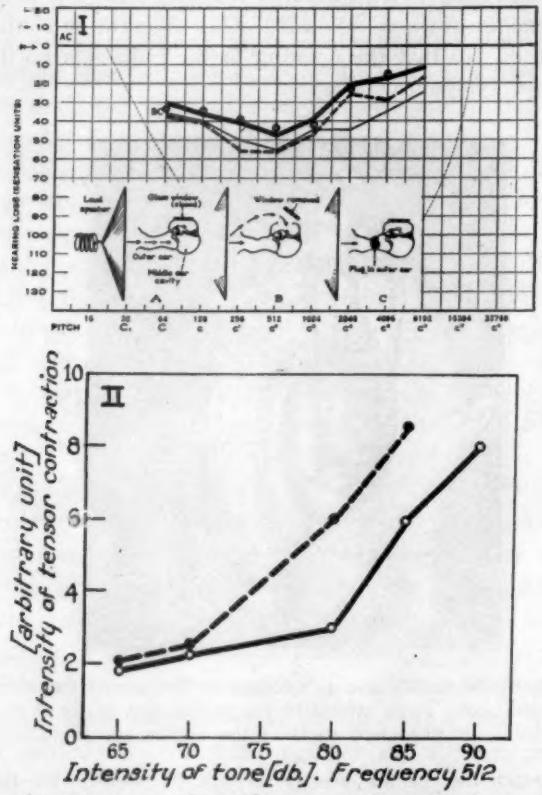

Fig. 2.—*I*, functional tests on the rabbit ear made under three experimental conditions. Under condition *A* the sound reaches the inner ear through the air of the outer auditory meatus, the ear drum and the ossicular chain. The threshold curve of the tensor tympani contractions is designated by the solid line. Under condition *B* the window of the middle ear has been opened. The threshold is practically unchanged. The threshold values are designated by a line with circles. Condition *C* demonstrates the effect of a cotton plug in the outer ear, resulting in a rise in the threshold of about 10 to 15 decibels (broken line).

II, threshold curves of the tensor tympani reflex contractions in the rabbit due to tones of different intensities. With an optical recording device the contractions of the tensor tympani in a rabbit are recorded on a screen. The abscissa represents variations of from 65 to 90 decibels in the intensity of the tone of 512 double vibrations. The unit of the ordinate is the excursion of the myograph on the screen. The solid line represents the test with the tympanic window closed (condition *A*, in *I*). The broken line designates the condition with the window open. Overthreshold intensities show that the reflex contractions are greater when the window is open than when it is closed. The area between the two curves represents the effect of aerocochlear conduction.

AEROCOCHLEAR SOUND CONDUCTION IN EXPERIMENTAL DEAFNESS

As an example of the simplest form of conduction deafness we have utilized the cerumen plug in the outer auditory meatus. Threshold tests were carried out on rabbits in which this condition was imitated by the insertion of wet cotton and boric acid ointment in the outer ear. Care was taken that there was no direct pressure against the membrana tympani. In addition, a fine glass tube was carried through the plug in order to equalize any pressure in the space between the plug and the ear drum. Occlusion of the outer canal in this way raised the threshold curve. If the bulla was then opened the threshold was again lowered.

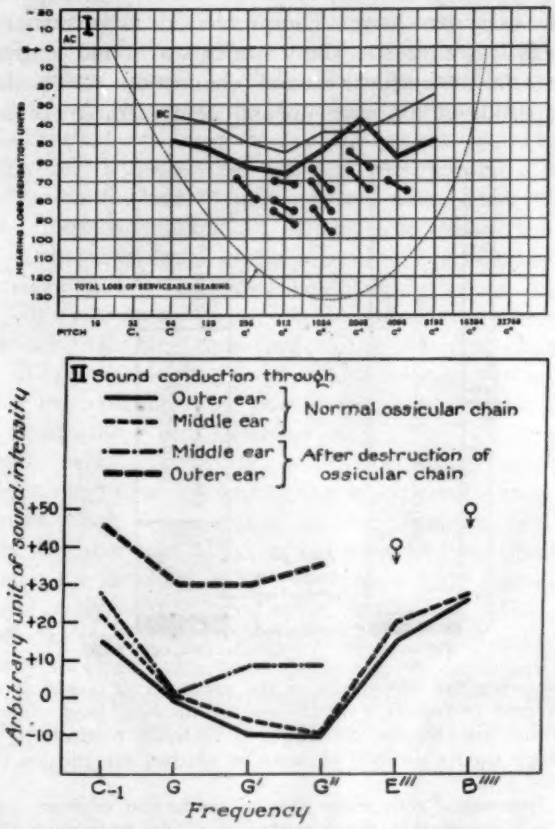

Fig. 3.—*I*, threshold and overthreshold tests. The solid line represents the normal threshold of the tensor tympani muscle of a rabbit. The oblique bars represent overthreshold intensities of equal contraction value. The left dot on each bar represents the contraction with the tympanic window open. The right end point designates the intensity which gives an equally strong contraction after the window has been closed. The intensity of the tone is raised until the contraction of the tensor tympani muscle equals that in the first test.

Note: The slant of the bars increases with intensity. This demonstrates that at stronger intensities the percentage of paraossicular conduction is increased.

II, aerocochlear sound conduction demonstrated after destruction of the ossicular chain. The threshold curve for the conduction of sound through the outer canal and that by way of the middle ear through the opening in the bulla are given. They are practically the same. After destruction of parts of the ossicular chain severe deafness results, as seen by the rise in threshold. If the window of the middle ear is opened the threshold is lowered.

In another series of experiments a number of perforations were artificially produced in the ear drum and the effect on the function was determined. Small

and medium-sized perforations of the drum were observed to be compatible both with fairly good ossicular sound conduction and with aerocochlear conduction.

Figure 3 *II* shows the effect of destruction of the ossicular chain on aerocochlear sound conduction. The threshold for sound reaching the ear by way of the external auditory canal was greatly raised. The threshold for sound entering by way of the open bulla was also raised, but to a lesser degree.

COMMENT

This paper deals with the problem of sound conduction through the air of the middle ear. Previously, anatomic and physiologic studies have, for obvious reasons, dealt mostly with the ossicular chain. The problem of nonossicular air conduction has found considerably less interest. Many students of sound conduction may even doubt the existence of aerocochlear sound conduction. For this reason, the emphasis in this paper is on the experimental demonstration of nonossicular air

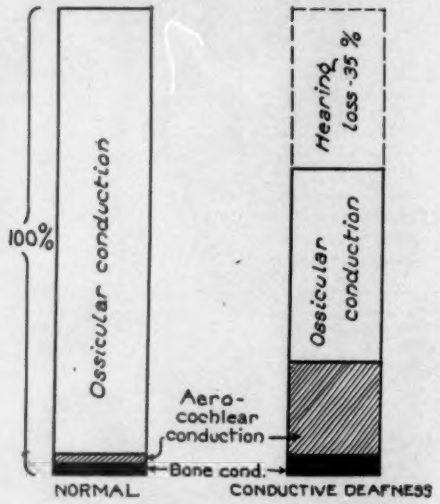

Fig. 4.—Scheme showing the importance of the pathways of sound at different levels of intensity. The left column represents a normal ear at threshold level. The large white part of the column shows that the ossicular conduction is the main conductor. Only a small percentage of sound energy travels directly by bone or reaches the cochlea through the air of the middle ear cavity.

The right column represents a theoretic case of conduction deafness with a hearing loss of 35 per cent. It is assumed that the contribution of the remaining 65 per cent is now different from the normal. The aerocochlear pathway is more effective and therefore has overcome partially the impaired ossicular conduction. There is also a small real increase in bone conduction.

conduction. Admittedly, a number of physical problems and exact quantitative calculations remain undiscussed.

From the animal experiments described in this paper it can be assumed that the relative importance of the pathways of sound varies with the intensity level. Experiments confined to threshold levels could not demonstrate the aerocochlear conduction. At higher levels of intensity the part of aerocochlear conduction becomes more definite.

From the study on the acoustic reflex of the muscles of the middle ear we have previously come to the following conclusions: The elastic qualities of the ossicular chain undergo characteristic changes due to the contractions of the muscles of the

middle ear. The intensity and the frequency of the sound govern these changes. A tone of low intensity does not cause a muscle contraction. Stronger tones produce tetanic muscle contractions and diminish the vibrating movements of the ossicles. In other words, a sound of underthreshold intensity for the muscle contraction is carried over the system with higher sensitivity than a louder tone which causes contraction of the middle ear muscles.

For overthreshold intensities the ear is "deafened" by the dampening effect of the middle ear muscles. This dampening effect, however, works mainly on the ossicular chain. The ossicular conduction, therefore, is mainly affected. Non-ossicular air conduction is not as much altered. The relative importance of nonossicular air conduction is therefore enhanced, as can be demonstrated experimentally. Similar observations can be made when the middle ear is deafened by experimental alterations. Figure 3 *II* demonstrates the destruction of the ossicular chain. In the presence of experimental deafness aerocochlear sound conduction is impaired also, but the relative importance of the various sound-conducting pathways has changed. Figure 4 is a schematic figure to illustrate this condition. We believe that in a normal animal the bulk of air-conducted sound energy is conveyed over the ossicular chain. Bone conduction and aerocochlear conduction are only negligible by-pathways. In the right hand graph a condition is demonstrated in which the animal has been deafened by an experimental lesion producing a conduction loss of 35 decibels. It is probably incorrect to assume that the experimental lesion impaired the sound-conducting capacities of the ossicular chain by an exact loss of 35 decibels. It is probable that the resulting function represents the conducting loss on the part of the ossicular chain plus partial compensation due to the increased effectiveness of aerocochlear sound conduction.

Additional information is urgently needed in order to understand more clearly the pathologic physiology of sound conduction in various types of deafness. Modern surgical procedures which aim to restore auditory function deal with conditions in the ear in which the ossicular chain is not functioning. Some operations even purposely interrupt the ossicles. These operations offer valuable material for the physiologist in the study of pathologic sound conduction. However, it is believed that, in addition to the clinical observations, a great amount of experimental research is necessary to give a clear physiologic understanding to empiric surgery.

The aforedescribed experiments provide evidence that in pathologic conditions the auditory function is not adequately expressed by determination of threshold.

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TREATMENT OF RETROAURICULAR FISTULA

REPORT OF FIVE CONSECUTIVE CASES

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The all too great incidence and the inherent difficulty of treatment of postauricular fistula make the subject one of prime importance to the otologist. Inasmuch as the success of any operative repair of this condition depends on complete and careful revision of the original mastoidectomy cavity as the first step, the problem is in the realm of otology rather than of plastic surgery.

This paper will concern itself with a brief review of the general aspects of the subject and a more detailed description of the method I have successfully employed in treatment in 5 consecutive cases. I shall not attempt to cover thoroughly the entire literature. The very fact that so many methods of therapy have been suggested is testimony that no one has yet met with complete and constant success.

There is little doubt that in many cases postauricular fistula can be prevented by the most careful and assiduous care at the time of the original mastoidectomy. The old adage "an ounce of prevention is worth a pound of cure" holds as true for postauricular fistula as for other postoperative complications. Any busy practicing otologist, however, remembers all too many cases in which the postauricular wound failed to close and yet the original operation had been performed with the greatest thoroughness. These cases must be placed in the idiopathic group. I can only suggest theories as to the reasons for their occurrence.

Careful technic should consist in complete removal of all areas of pathologic tissue, all granulations and all diseased bone. This is particularly true in the radical mastoidectomy; in this operation all cholesteatomatous tissue should be followed to its source and completely exenterated, along with all overhanging bone. Cholesteatomatous tissue is often extensive. It is not infrequent to encounter a cholesteatoma burrowing from the antrum along the facial ridge to the tip of the mastoid bone, around the sinus plate, along the tegmen tympani.

Therefore, in the presence of a sclerotic mastoid with a cholesteatoma I should not consider it sufficient surgical treatment to perform an antrotomy and clean out the cholesteatoma and the tympanic cavity. All the bone down to and including the tip of the mastoid bone and extending over the sinus plate should be carefully removed. It is all too common, at the time of operation, to discover a diseased bone cell behind the sinus plate and at the tip of the mastoid which roentgenographic examination had previously failed to reveal. In addition to thorough excision as a preventive measure, I believe that suturing that coaptates the edges of the skin without tension and the avoidance of strong antiseptics, which devitalize the tissues and rob them of healing properties, are greatly worth while.

The formation of postauricular fistula is fairly typical in its course and pathologic picture. For some reason the wound fails to close. The sutures slough, and granulations do not fill the defect from below. The edges of the skin gape, become devitalized and invert, with formation of a fistulous tract down to the bone, which is usually covered with infected tissue, from which exudes a variable amount of

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annoying discharge. There may or may not be concomitant pain. At any rate, the pain or discomfort is never a prominent feature. The tympanum also is seen to undergo changes. The usual condition encountered is a dry middle ear with a thickened drum membrane. The existence of a postauricular fistula, per se, seems to affect hearing only slightly, if at all.

Retroauricular fistula is seen about equally in males and in females, and the age and the general condition of the patient do not seem to be factors. Greenfield,[1] indeed, in his report of 8 cases, stated that the failure to heal occurs "oftentimes in children who have good care, live in excellent hygienic surroundings and are well fed and well nourished."

Formerly, many pioneers in the field of radical mastoidectomy considered a permanent postauricular opening to be essential. They expressed the belief that such a fistula is beneficial for the aeration of the radical mastoidectomy and the tympanic cavity. Eagleton[2] stated: "If it were not for cosmetic purposes these fistulae should be left alone." I do not entirely disagree with this attitude, and thus I believe that at least one year should be allowed to lapse before surgical intervention is carried out on a retroauricular fistula following radical mastoidectomy. Indications for operation, aside from the cosmetic one, include eradication of foci of infection, changes in the middle ear, existence of an unsightly and annoying discharge and the possible loss of hearing.

Retroauricular fistula has been treated in various ways. Medical treatment is seldom successful and really holds no place in the therapy except in the case of a very small sinus. Numerous means have been employed to fill in the defect surgically and close the wound. Eagleton[2] first used bone grafts and chips in the reconstruction of the cavity. Copps and McCormick[3] had good results with not too extensive defects by using subcutaneous pedunculated flaps. Straatsma and Peer[4] claimed not only that they obtained satisfactory closure with the use of free fat grafts but that it was a prompt method of filling the defect and stimulated healing by the formation of new capillaries. Popper[5] suggested the use of periosteal flap grafts routinely in the mastoid operation.

Thus it can readily be seen that the successful correction of postauricular defects depends on the use of various types of tissue grafts and flaps. Simple undermining of the skin adjacent to the fistula with secondary closure often is sufficient in the case of small, clean defects if it can be completed without the skin's being placed under too great tension. A modification of this simple method is the use of a purse-string suture employed by Passow for closing the deep layer of a double layer flap made from adjoining tissue.

The simple epidermal flap is excised from below the defect, an attachment being left inferiorly, and placed with epithelium first into the cavity. Modifications, with or without relaxing incisions, have been described by Beek, Goldstein, Trautmann and Watson.[6]

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Regardless of the type of operation chosen, there are two pathologic foundations for the persistence of a fistula. There is always present a sequestrum, which acts as a foreign body and which must be eliminated before satisfactory closure can be obtained. Also, the soft tissues have become devitalized and atrophied and are not suitable for use as building material in the closure of the defect. Therefore some outside healthy tissue must be employed.

Greenfield [1] noted breakdown of repair in 2 cases in which he failed to open and clean out the mastoid cavity; healing followed completed revision. From my experience, the combination of the sliding flap and temporal muscle graft in cases in which the fistula is large and of long duration, with thin, unhealthy surrounding skin, yields the best results. This is especially true in cases in which previous attempts at closure have been made. The use of the temporal muscle flap is not new, but the recent refinement of technic brings a high percentage of cures.

The details of the method I have successfully employed in 5 consecutive cases are outlined in the following discussion.

Fig. 1.—Two incisions employed.

DESCRIPTION OF METHOD

Preparation of Operative Area.—1. The entire half of the scalp is carefully shaved. 2. The area is prepared with tincture of green soap, alcohol, ether and metaphen. Metaphen is the antiseptic of choice because of its high germicidal properties and low tissue-devitalizing qualities.

Anesthesia.—General anesthesia should be employed. Local infiltration distorts the tissues and further embarrasses an already poor circulation. The type of general anesthesia, of course, depends on the individual patient and the coexistence of other pathologic conditions.

Incision.—Incision is made through the old scar down to the bone, or the old scar is excised, as much surrounding tissue being left as possible.

Revision of the Mastoid Cavity.—All epithelium lining the fistulous tract should be removed. In the elevation of the soft tissues the periosteum is carried along with them. The best instrument for this purpose I find to be the Freer elevator.

The anterior flap is retracted as far forward as possible; this gives maximal exposure to the zygomatic area and brings the posterior canal wall into view. The posterior flap is also retracted well out of the way.

The entire mastoid area should now be carefully and completely revised. All granulation tissue, diseased bone and infected cells are diligently sought and removed with curet and rongeur. Too much stress cannot be placed on this stage, and unless it is complete, reinfection will follow, with resultant fistula.

The mastoid tip is now removed well above the digastric ridge, with exposure of the digastric muscle. The posterior canal wall is lowered, and granulations are removed from the antrum. The lowering of the posterior canal wall will help to diminish the size of the defect.

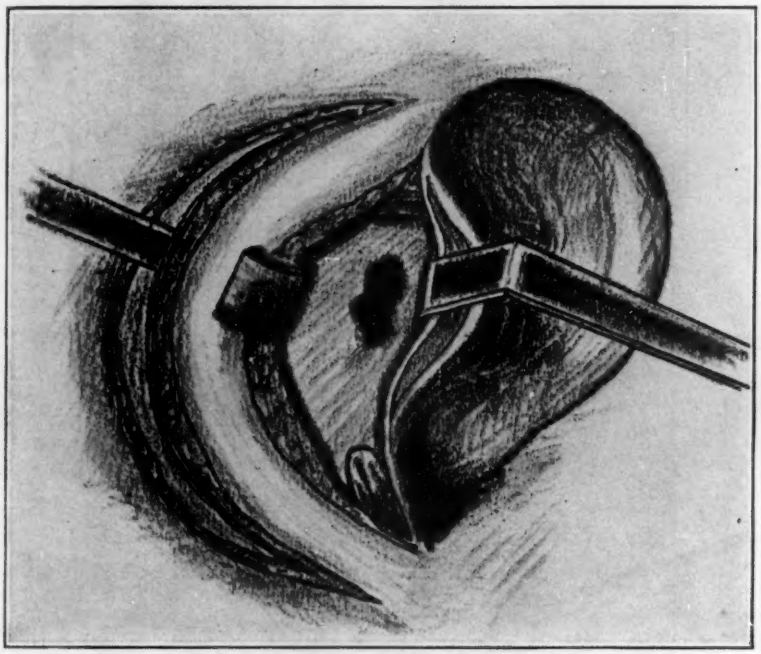

Fig. 2.—Appearance after formation of sliding flap, with fistula exposed.

Sliding Flap.—At this stage, a curved incision is made parallel with and about 1½ inches (3.8 cm.) posterior to the initial one. The flap is made by elevating the skin and periosteum between the two incisions; this is best accomplished by working from behind forward. It is essential that the flap be entirely free from the tip if it is to relieve successfully the tension of the sutures.

Temporal Muscle Flap.—The original mastoid incision is extended from its upper angle for a distance of about 2 inches (5 cm.), the incision being inclined slightly anteriorly through the skin only. Great care should be exercised not to cut into the temporal muscle or fascia, as it is here that bleeding is most profuse. Complete hemostasis is essential to the success of the repair. A hematoma, as is only too well known, will either break the wound open primarily, through physical tension, or secondarily, as a result of infection. Separation of the skin from the temporal fascia is best done with a pair of Mayo short curved scissors, which should hug the skin but not button-hole it.

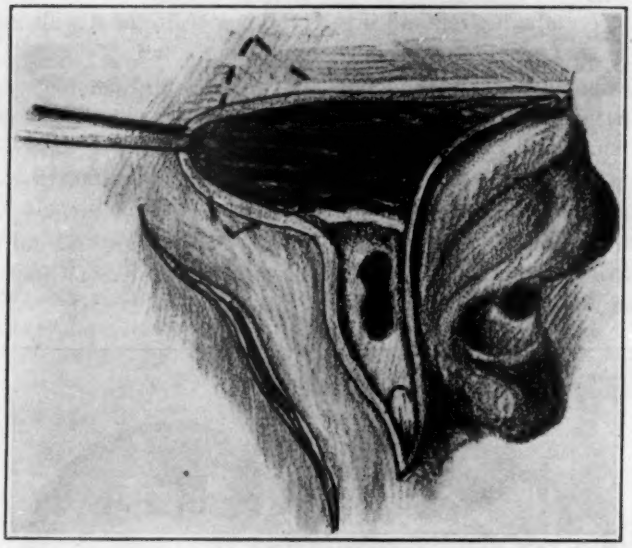

Fig. 3.—Fan-shaped section of the temporal muscle.

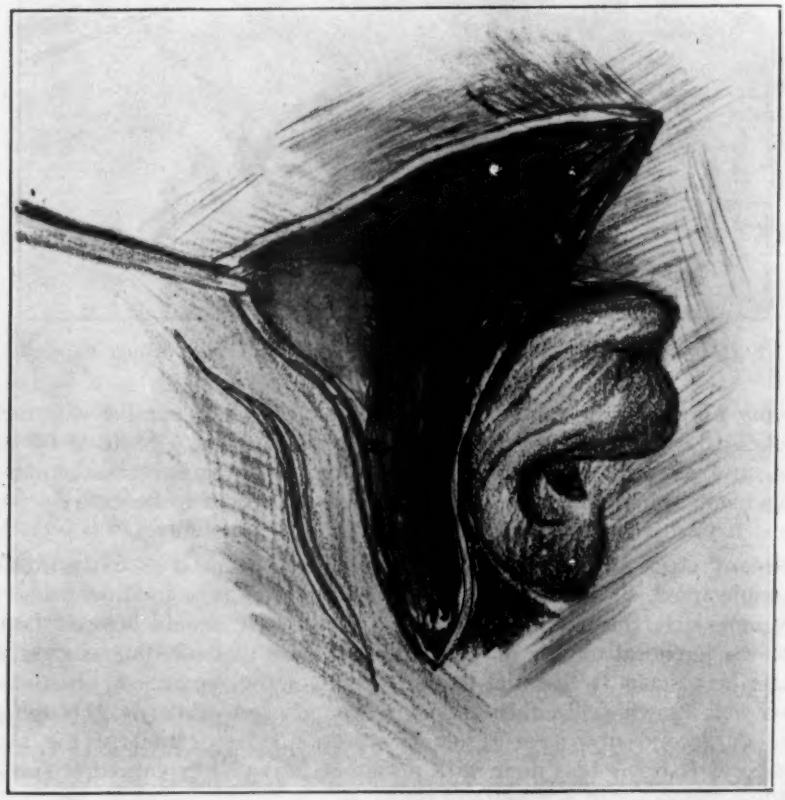

Fig. 4.—Muscle flap sutured to the digastric muscle, which fills in the mastoid cavity.

Hot pads saturated in saline solution are applied, and an incision is made just above the auditory canal, at right angles to it, along the attachment of the temporal muscle to the temporal crest and running posteriorly toward the occipital tubercle. This incision should extend through fascia, muscle and periosteum for a distance slightly longer than that from the tegmen to the tip of the mastoid.

After all bleeding points are ligated, the periosteum is freed from the bone for a distance of about 2 inches. The flap is now prepared, but before it is cut the mastoid area to be covered is studied. The flap should be cut slightly larger than the area to be filled, with allowance for contraction and atrophy. The flap should be shaped like a fan, with its pedicle at the auditory canal. It will usually be

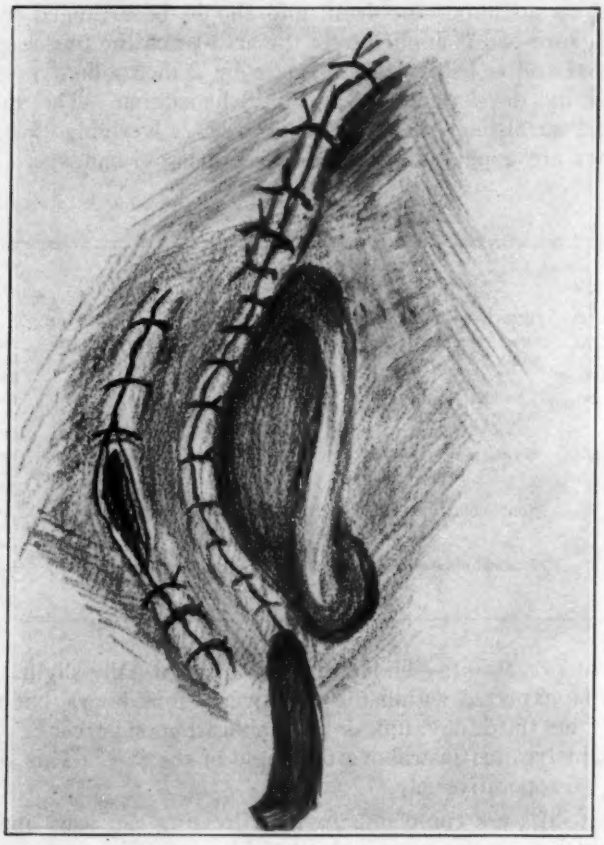

Fig. 5.—Method of closure.

found to measure about ¾ inch (1.8 cm.) in width. If the flap is not long enough to meet the digastric muscle without undue tension, it may be incised transversely across its fibers in one or more places. This will serve to lengthen the flap and to relieve tension.

The temporal flap is next sutured to the digastric muscle, it being borne in mind that a large artery, the occipital, lies just beneath the digastric muscle. Chromic no. 1 catgut is the suture of preference, and one or two interrupted sutures are usually sufficient. The flap is secured to the surrounding soft tissues with two or three additional sutures. The mastoid area is now completely covered with a good arterial flap, which will act as a firm support for the overlying skin.

Suturing of the Flap.—The skin at the auricle is next undermined and sutured to the sliding skin flap after the edges have been freshened. Dermal or silkworm sutures, extending through the skin and periosteum, are employed. The skin posterior to the second incision is undermined and sutured either partially or in its entirety. If there is any tension whatever, it is best to allow the edges to remain apart and to apply a petrolatum gauze pad. A small rubber drain is inserted at the lower angle of the mastoidectomy wound.

Dressing.—The entire operative area is inspected and sponged dry. Hydrogen peroxide is a suitable agent for the clensing of the skin and auricle. No stronger antiseptic should be used, as it tends to devitalize the skin, and it is especially important that the mastoid incision heal by primary union.

The dressing is an important detail and should be arranged by the operator. A thick, 2 inch gauze pad is applied over the area when the muscle-periosteum flap has been removed and is held firmly in place by 2 inch adhesive tape. This will serve to prevent the development of a painful hematoma. The auditory canal is sponged dry and an alcohol gauze strip inserted. Overlying this area abundant gauze compresses are applied and secured by a mastoid bandage.

Data on Five Consecutive Cases

Case	Sex	Age, Yr.	Duration of Defect	Previous Operations	Previous Attempts at Repair	Impairment of Hearing	Complications	Result	Operation
1	F	15	7 yr.	One simple mastoidectomy	None	None	None	Cure	Revised simple mastoidectomy and temporal flap
2	F	29	5 mo.	Two simple and one revision of radical mastoidectomy	One	None	None	Cure	Revised radical mastoidectomy and temporal flap
3	F	38	10 mo.	Simple mastoidectomy	None	None	None	Cure	Revised simple mastoidectomy and temporal flap
4	M	38	8 mo.	Simple mastoidectomy	None	None	None	Cure	Revised simple mastoidectomy and temporal flap
5	F	37	22 yr.	Radical mastoidectomy	None	None	Dropping of temporal flap	Cure	Revised radical mastoidectomy and temporal flap

Postoperative Treatment.—The febrile reaction is usually slight. A temperature of 101 F. is to be expected within the first twenty-four hours, but there is usually no fever after the third day unless a complication supervenes. Most patients complain of slight trismus just above the angle of the jaw. This sensation is rare after the tenth postoperative day.

If the patient does not complain, the first dressing can wait until the fourth or fifth day, at which time the area is dressed like any other surgical wound. The sutures are removed at the second dressing, and the drain is removed as soon as the canal is dry and the drainage from the mastoid cavity is slight. If convalescence is uneventful, all dressings may be removed at the end of two weeks.

From the accompanying table it can be seen that in these 5 cases the age varies from 15 to 38 years and the fistulas had been present from five months to twenty-two years; the fistula followed a simple mastoidectomy in 3 cases and a radical mastoidectomy in 2 cases. There was one complication, slipping of the flap into the tympanic cavity. Operation in all cases resulted in cure.

If a radical mastoidectomy is contemplated, the same plastic closure may be utilized, but, I think, a Siebenmann plastic flap is advisable. In this operation the

temporal muscle-periosteum flap is placed on top of the upper and lower legs of the Siebenmann flap. As a precaution, the muscle flap may be caught with two of the skin sutures to prevent it from slipping into the tympanic cavity.

In conclusion, I wish to stress that I have found this operation to be the most suitable in cases of large retroauricular fistula, in cases in which the fistula is of long standing and in cases in which previous attempts at repair have been made.

Close attention to detail is imperative. Complete asepsis, thorough preparation of the field of operation, complete revision of the mastoid area, careful handling of the tissues and capable after-care should result in a complete cure.

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EXACT DIAGNOSIS OF OTITIS MEDIA

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It is generally agreed that surgical incision of the tympanic membrane does no harm and a "dry tap" is more excusable than failure to provide drainage as soon as needed, but pride in diagnostic skill is always embarrassed by fruitless myringotomy and sometimes the ear starts discharging one or more days after incision, a complication suggesting that the insult to continuity of tissues and their circulation may have led to suppuration otherwise avoidable. Moreover, the sulfonamide compounds today offer a fairly rapidly effective and specific agent with which to combat the common invaders of the tympanic cavity and to abort infections which have not reached a stage requiring drainage.

Semenov,[1] Fowler,[2] Fenton,[3] Lierle and Potter [4] and Polvogt and Bordley [5] have clearly demonstrated that inflammation in the middle ear differs no whit from that in the same types of tissue elsewhere and that it passes through the same stages. The stages of inflammation are (1) congestion, (2) edema, (3) cellular exudation and (4) necrosis.

1. Congestion is first "active," or arterial; i. e., it is due to dilatation on the arterial side of the capillary bed, with an increased blood flow to the area—seen by the otologist as a "bright red" membrana tympani. Later congestion becomes "passive," as a result of dilatation on the venous side, so that stasis is added to the increased vascularity, producing the dull, "rosy" or dark to purplish red drum membrane. Congestion, no matter how extreme, is entirely self resolving provided the inflammation goes no farther.

2. Edema, the intercellular extravasation of fluid, needs no description. In the ear it is largely responsible for obliteration of landmarks and thickening of the membranes and may impede drainage. It, too, is self resolving, though collections of transudate in body cavities may best be evacuated conservatively. In the ear this can usually be done by inflations.

3. Cellular exudation consists of the diapedesis of mononuclear and polynuclear hematopoietic leukocytes, the activation of fixed tissue phagocytes and the stimulation of fibroblastic proliferation. It adds to the bulk and thickening of the tissues and varies from scattered infiltration, sometimes with miliary interstitial collections forming microscopic abscesses, to massive invasion, even to the point of disruption of tissues. That the development of pus-filled cavities or spaces requires surgical drainage to assure recovery is still a first surgical principle.

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4. Necrosis is, of course, fatal to the tissues involved and, if not arrested, to the organism as a whole. It tends to advance so long as drainage is impeded. Its repair always produces some loss of function, proportional to the extent and site of the damage. Surgical methods of control vary from simple drainage to radical excision of the involved areas. In the ear necrosis varies from spontaneous perforation, which is actually a process of necrosis, and not a pressure effect, through more or less permanent destruction of functioning structures of the tympanum, on to mastoiditis and its complications.

These stages are not, of course, sharply defined but overlap throughout. For instance, microscopically demonstrable leukocytic activity, and even focal necrosis, occurs when the third and fourth stages of inflammation cannot be diagnosed clinically, and long-standing or repeated inflammations which do not pass grossly beyond the congestive stage stimulate fibroblastic proliferation and ultimate scarring, and irreversible injury to hearing may ensue. Nevertheless, in otologic practice a sufficiently accurate diagnosis can be made, and the prompt recognition of the development of the third stage (suppuration) and the provision of surgical drainage thereby indicated are highly important. A clear concept of these stages is helpful in this performance, and conscious noting of the rapidity with which each stage succeeds the other gives a rough but useful estimate of the ratio of virulence of the invading organism to the resistance of the host, and thus aids in predicting what the subsequent course is apt to be.

Diagnosis consists in making observations, analyzing the facts obtained and drawing conclusions as to what has occurred, what will probably follow and what steps should be taken to promote optimum recovery. The observations from which the diagnosis of inflammation can be made, regardless of the site, are the "cardinal signs of inflammation," viz., redness (rubor), heat (calor), pain (dolor), swelling (tumor) and loss of function (functio laesa).

In otitis media most of these signs are highly fallible. 1. Redness, the result of the congestive stage, varies remarkably and is altered by the development of later stages. It is unnecessary to tell otologists that many very red ear drums do not need incision or that many others do when they are hardly more than pink or salmon red or are red only in part of the quadrants. 2. Local "heat" is not observable by any practical means available at present, even if it would be useful to know, and general elevation of temperature is even more fallible than redness. 3. Pain is not dependable, either by its presence or by its absence, as a determining factor on which to decide the diagnosis, prognosis and treatment. 4. Swelling consisting only of edema is self resolving, but it often resembles "bulging" due to pus under pressure. The latter form of swelling is the most commonly accepted criterion of suppuration warranting surgical drainage, but it is not mensurable, and experienced otologists are often puzzled, and many times their judgments differ, as to whether in an individual case there is "actual," or "enough," bulging to warrant incision. 5. Loss of function is not mentioned in otologic texts or in the literature except as a comment that it occurs. This is strange, as it is the only one of the five cardinal signs that is accurately mensurable, the otologist being peculiarly fortunate in this. It is my present thesis that measurement of loss of hearing provides a definite, simple and infallible sign by which the stage of advancement of inflammation in the middle ear can be diagnosed.

Chancing to test the hearing in a case about ten years ago when I was quite put to it whether to incise or not, I was surprised in the presence of the other signs to an intense degree to obtain a positive Rinne reaction. I procrastinated, and rapid resolution followed. I thereon began to test hearing in more cases of acute otitis media and am now prepared to state unequivocally that no patient, regardless of the

presence of redness, pain, fever or apparent bulging of the tympanic membrane, needs paracentesis so long as air conduction remains better than bone conduction. This rule has not failed me in the past eight years and in over 100 cases has enabled me confidently to avoid surgical intervention when I should otherwise have feared to delay it. Typical case histories follow.

Mrs. M. had had a sore throat for the past week and severe earache for the past twelve hours. An attack of acute follicular tonsillitis was subsiding. The right membrana tympani was covered with hemorrhagic vesicles; no landmarks were discernible, and the entire area was bulging. The temperature was 101 F. Some relief of pain was obtained from pricking and emptying the vesicles with an applicator. The Rinne reaction was positive. Radiant heat was applied. Two days later inflammation in the drum had largely resolved and landmarks were distinct. Uninterrupted recovery followed.

R. B., aged 8 years, had had an acute infection of the upper respiratory tract for the past four days and severe pain in the ear for several hours. The temperature was 101.8 F. The left drum membrane was fiery red and appeared to bulge. Landmarks were not distinguishable. Several small serous vesicles were noted in the anterior and superior quadrants. The Rinne reaction was positive. Application of radiant heat was comforting, and definite resolution was demonstrable next day.

I. J., aged 4 years, had had an acute infection of the upper respiratory tract for the past week and a vague earache for the past two days. The temperature was 100.2 F. The tympanic membrane were dusky red, swollen or bulging. Landmarks were identifiable but obscured. The Rinne reaction was definitely positive. Resolution followed conservative care.

COMMENT

It is surprising how easily very young children, if at all cooperative, can be tested. They are used to listening to father's watch as a game, and after a few repetitions a satisfactory estimate of the Rinne test can be obtained. The otologist can also calibrate his watch to that distance at which the Rinne reaction becomes negative in other patients with normal bone conduction and use this calculation effectively. Or he may compare a tiny patient's air conduction with his own bone conduction with sufficiently approximate results.

The almost universal rule that negative results are never as significant as positive ones holds good here, and a negative Rinne reaction does not mean that incision of the tympanic membrane is necessary unless other results are in accord. The possibility of preexisting damage to the middle ear with a permanently negative Rinne reaction must be kept in mind, and a patient with nonsuppurative "serous" or "catarrhal" otitis can manifest a negative Rinne reaction. Bone conduction must also be normal.

It cannot, of course, be assumed that a positive Rinne reaction will remain so in the presence of acute inflammation, and adequately close observation must be maintained if paracentesis is postponed on the strength of this sign alone.

SUMMARY AND CONCLUSIONS

Otitis media passes through the same four stages as do other inflammatory processes. When the stage of cellular exudation has produced a tympanic empyema, surgical drainage is indicated, but not before. Of the cardinal signs by which inflammation of the middle ear can be diagnosed, loss of hearing (functio laesa) is the only definitely mensurable one. A positive Rinne reaction provides an infallible sign that the third (suppurative) stage of inflammation has not appeared.

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LYMPHOEPITHELIOMA OF THE NASOPHARYNX

REPORT OF A CASE

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The following case is presented because of a number of unusual features.

REPORT OF CASE

Mrs. C. M., aged 52, was first admitted to the surgical wards of the Mount Sinai Hospital on July 17, 1940 with a complaint of a lump on her neck. This tumor was first noticed thirty years before, and she dated its occurrence to an injury which she suffered when she fell from a horse. Small at first, it became progressively larger, until the time of admission it was the size of an orange. The growth was always symptomless, freely movable and painless and moved up and down with swallowing. Her past medical history did not bear any relation to this tumor, although she had exhibited both obesity and hypertensive cardiovascular disease for many years. In the course of physical examination it was noted that her right tonsil was greatly hypertrophied, extending beyond the midline, and was rather firm but painless. There were no symptoms referable to interference in swallowing, but there was a sensation of fulness from time to time. There was no evidence of any acute infection. The left tonsil, though cryptic, was normal in size. On the right side there was a palpable, painless submaxillary lymph gland.

The provisional diagnosis on admission was (1) thyroglossal cyst, (2) colloid goiter, (3) pronounced hypertrophy of the right tonsil, possibly malignant (lymphoepithelioma or lymphosarcoma[?]), (4) hypertensive cardiovascular disease and (5) obesity.

Examination of the nose and throat the following day revealed the presence of pronounced hypertrophy of the right tonsil, with a small superficial ulceration on its mesial surface. The tonsil was firm to touch, and seemed to fill the entire tonsillar fossa. It extended upward above the level of the soft palate and downward along the carotid artery. The impression at the time of examination was that of a malignant condition of the tonsil—a mixed tumor, such as seen in the parotid gland, a lymphosarcoma or some similar lesion.

On July 24 the right tonsil was removed with local anesthesia. The mass was observed to be well encapsulated, was enucleated from the fossa readily and did not show any residual masses. The remaining capsule was fibrotic and did not show any gross evidence of a break due to possible infiltration of the tumor tissue. There was no evidence of extension of this tissue along the large vessels of the neck, nor were the cervical glands palpable. At the time of the operation, there was no undue bleeding, and the postoperative course was uneventful. The specimen was sent to the laboratory for pathologic study.

A week later, on August 1, a large cystic adenoma of the thyroid was removed, with local anesthesia. Again, the postoperative course was uneventful, and the patient was discharged from the hospital on August 7, definitely improved.

The pathologic report on the tonsil revealed that it was 2.5 by 2 cm. in size and did not show any gross evidence of a malignant process. Microscopically, differentiation and identification were rather difficult. The specimen was covered by a layer of stratified squamous epithelium, under which hyperplastic lymphoid tissue was undergoing fibrosis. Some areas showed collections of moderately pleomorphic cells, the dominant cell being rather large, with a prominent, heavily stained nucleus. There was some mitotic activity in the larger hyperchromatic cells. The impression was that the condition might be early Hodgkin's disease or a lymphoepithelioma, but the diagnosis was not conclusive (fig. 1).

After the patient's discharge from the hospital, she was in rather poor physical condition, owing to a number of episodes, which included an attack of cardiac decompensation, arthritis and hypertension. She was hospitalized for various periods and apparently had responded favorably to treatment.

From the otorhinologic service no. 3, Mount Sinai Hospital.

On June 9, 1941 she returned to the outpatient department of Mount Sinai Hospital, primarily because of nasal obstruction and a noticeable swelling along the right side of her neck. This swelling had been noticed by her daughter four months before. Small at first, it gradually became larger, but was always painless and apparently symptomless. Casual examination of the nose revealed a moderately congested mucous membrane, a small amount of mucoid discharge in both nostrils and definite limitation to free breathing. This inspection revealed no polypi or other evidences of a pathologic process. Examination of the throat showed a clean right fossa, without any evidence of recurrence of the original tumor. However, along the right side of her neck the entire chain of cervical lymph nodes was palpable and enlarged. Each node was discrete, freely movable and painless.

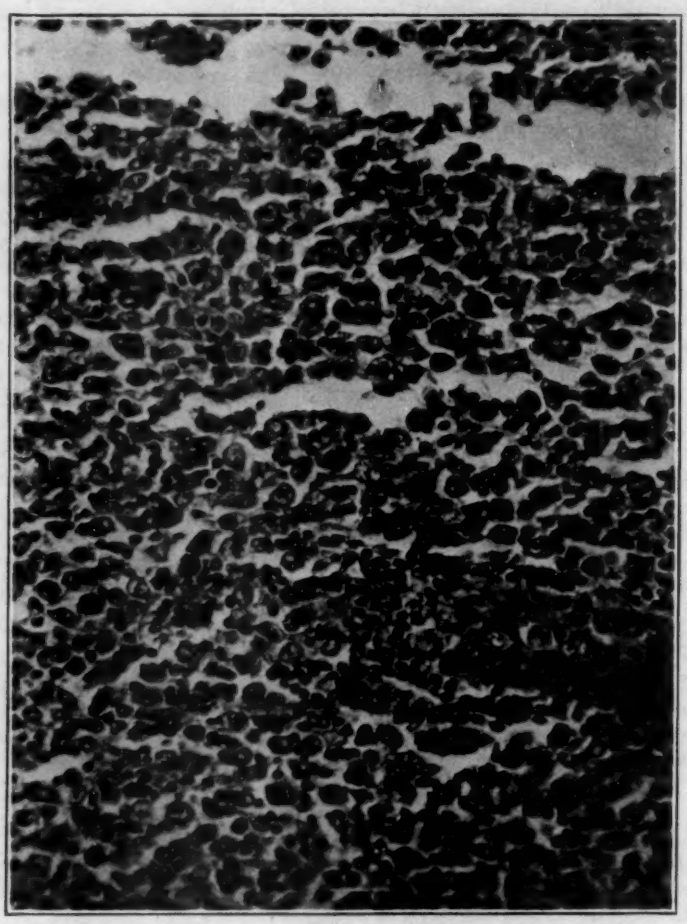

Fig. 1.—Photomicrograph of the tonsil; × 350. Many pleomorphic cells, some large, with prominent, heavily stained nuclei, are present. Active mitosis is evident in some of the large hyperchromatic cells. A moderate amount of fibrosis is noted throughout.

In view of the previous history and the aforementioned observations, a tentative diagnosis of Hodgkin's disease was made, and the following day the patient was readmitted to the ward, where routine studies were begun. Biopsy of one of the cervical glands was contemplated. While I was awaiting the results of laboratory tests and the roentgenographic examination of the chest, prior to performance of biopsy, the patient suddenly began to expectorate a small amount of blood. The following day she vomited a large quantity of black blood. Careful rhinoscopic examination showed that the bleeding came from a large, soft, well encapsulated mass high in the nasopharynx, well above the level of the soft palate. Manipulation of or contact with the mass produced free bleeding. On June 17 several pieces of

tumor tissue were punched out for biopsy. The patient was then exposed to high voltage roentgen irradiation. The following day the bleeding stopped, but the roentgen exposure was continued daily for several weeks. The tumor regressed rapidly, so much so that when the patient was discharged, on June 29, it was impossible to find any residual evidence of tumor tissue, and locally her condition could be considered cured.

The pathologic diagnosis of the tissue removed at biopsy, again, was rather inconclusive (fig. 2). "The tissue was densely cellular. Most of the cells were small and heavily stained; other cells were smaller. A few larger cells, some with multiple nuclei, were also present. Considerable fibrosis was proceeding in many areas. The diagnosis of Schmincke's tumor (lymphoepithelioma) was strongly suggested."

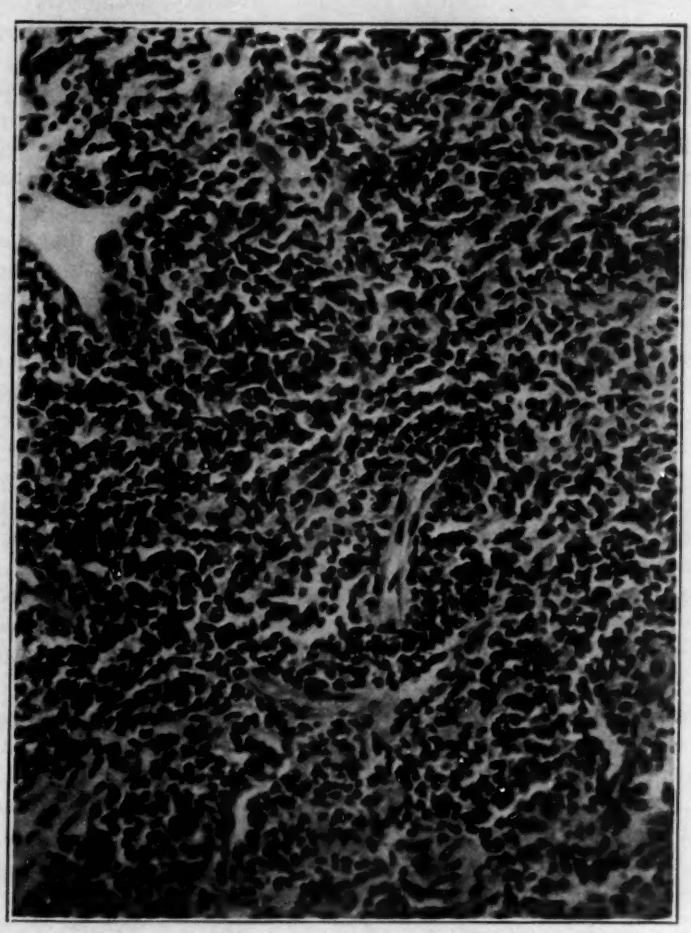

Fig. 2.—Photomicrograph of tissue from the nasopharynx; × 350. The cells are somewhat smaller. Hyperchromia is present. Many cells have multiple nuclei. Fibrosis is more extensive and general.

Shortly after her discharge from the hospital her hypertensive cardiovascular condition had again become a factor confining her to bed for various periods, until on Nov. 5, 1941, she died suddenly of a coronary occlusion.

COMMENT

This case presented two rather interesting features—one, painless symptomless enlargement of the right tonsil, which on biopsy revealed malignant hypertrophy, of a type which presented marked difficulty in diagnosis, and second, one year later,

a nasopharyngeal malignant growth, suggestive of lymphoepithelioma. The latter lesion was characterized by nasal obstruction, free bleeding, radiosensitivity and histologic peculiarities, observed at biopsy.

The question to be settled is the correlation of the two pathologic processes. Is the nasopharyngeal growth merely a recurrence or a metastasis of the original tumor in the tonsil? Are the two malignant growths of a somewhat different nature and origin, occurring about a year apart? There is much similarity between the two growths; yet there is much that is contradictory.

Neoplasms of the tonsil, both primary and secondary, are not infrequent. The primary benign tumors include the leukemias and the fibroma, fibrolipoma, angiofibroma, angioma, psammoma, mixed tumors of the salivary type, reticuloma, papilloma and dermoid cyst. The malignant tumors include the lymphosarcoma, lymphoepithelioma, transitional cell carcinoma and squamous cell carcinoma. The secondary tumors, other than those invading the tonsil by continuity, are metastatic from many remote sources, such as the breast, the lung and the intestine.

Our case presented unilateral enlargement of the tonsil. While grossly the tonsillar mass appeared fairly firm, well encapsulated, freely movable and slow growing, with only slight evidence of cervical adenopathy, microscopically it was definitely malignant. However, the sections revealed a picture difficult to classify because of the pleomorphic collections of cells—some large, some small, some hyperchromatic, with marked mitotic activity—and in other areas a great many lymphocytes undergoing fibrosis. Although there was some resemblance to early Hodgkin's disease, this was not conclusive. Lymphoepithelioma or a transitional cell carcinoma had to be ruled out. Clinically, however, there was no evidence of Hodgkin's disease.

One is now confronted with the second interesting feature. Nine months after her discharge from the hospital the patient noted slow, progressive enlargement of the cervical glands of the right side of the neck. This enlargement, again, was painless and symptomless and apparently did not interfere with muscular movements, such as talking, eating, swallowing or turning of the head. However, owing to incapacity caused by a flare-up of arthritis and secondary cardiac decompensation, three months elapsed before she returned to the clinic.

As previously noted, a diagnosis of lymphoepithelioma was made. The question arises as to the relation between the two pathologic processes. There have appeared many reports of cases of lymphoepithelioma since Regaud [1] and Schmincke,[2] working independently, described its characteristics. Ewing [3] stated that the tumor is the most characteristic neoplasm of the nasopharynx. He said:

> The chief locations are the posterior nares, the orifice of the eustachian tube, the vault of the pharynx and the tonsillar areas. The first symptom is generally an enlargement of the lymph nodes in the neck. . . . One or more years may elapse between the appearance of the cervical metastasis and the detection of the primary tumor.

Jovin [4] described lymphoepithelioma as a small, often silent, primary growth, progressing slowly, with either early or late metastasis and high radiosensitivity. It may occur at any time, but chiefly between the ages of 30 and 60 years. The location may be in any portion of the nasopharynx, but most frequently about the eustachian orifice, the posterior nares or the vault of the pharynx.

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1. Regaud, C., cited by Ewing.[3]
2. Schmincke, cited by Ewing.[3]
3. Ewing, J.: Neoplastic Disease, ed. 4, New York, W. B. Saunders Company, 1940, p. 785.
4. Jovin, J.: Lymphoepithelioma and Transitional Cell Epithelioma, Bull. Assoc. franç. p. l'étude du cancer **28**:117, 1939.

The tonsils, the vault of the pharynx, the nasal passages, the base of the tongue and the nasopharyngeal sinuses are covered in part by a stratified squamous epithelium, the cells of which are delicate, do not produce keratin and show little tendency to hornify. The mucous membrane is often interlaced with lymphoid tissue, which has been given the name of lymphoepithelium. Tumors arising from this tissue have peculiar clinical and histologic characteristics. They are often anaplastic and are more closely related to the squamous than to the basal cell carcinoma (Cloward [5]).

The origin of the tumor is a mooted question. One group of investigators believe that it is of branchiogenic origin and that it develops from remnants of the branchial arches and clefts, the islets of lymphoid tissue remaining dormant for many years, when suddenly cell proliferation occurs. On the other hand, there are many who believe that the source is purely epithelial and has no relation to the branchial clefts.

Furthermore, confusion exists with respect to the diagnosis of such a tumor. There are essentially three types of tissue in the pharyngeal region: squamous epithelium, transitional epithelium and the so-called lymphoepithelium. Often it is almost impossible to differentiate histologically between the last two types. The lymphoepithelioma appears as a tumor showing epithelial cells in wide sheets and cords, growing diffusely and infiltrating the surrounding lymphoid tissue. The cells are large, pale and delicate, with a thin cell membrane. The protoplasm is ill defined, has a trabeculated arrangement and often forms a true syncytium. The nuclei are large, clear and pale and show great variation in size. Nucleoli are prominent, and mitosis is abundant. In places the lymphocytic infiltration is so thick that the tumor cells are hardly visible through the lymphoid structure. Occasionally, one sees large cells in the reticulum—round or polyhedral, with hyperchromatic, and sometimes multiple, nuclei.

On the other hand, the transitional cell carcinoma consists of a mass of moderately large cells with indistinct cell borders. In some regions the cells are closely approximated, simulating epithelium. In others they are loosely arranged and resemble reticulum cells. The nuclei are pale and round or oval and hyperchromatic. The cytoplasm is moderate in amount, and the individual cells present wide differences in size and shape. Numerous mitotic figures are present. There is also a scattering of lymphocytes.

The similarity is so great histologically that a differential diagnosis is often difficult—so much so that many times a discordance of opinion appears when the same specimens are presented to different pathologists.

Clinically, the lymphoepithelioma appears as a slow-growing, infiltrating tumor, with a tendency to early metastasis, especially to the cervical lymph nodes, and is extremely radiosensitive. The principal symptoms—those of nasal obstruction, difficulty in breathing and obstruction of the eustachian tube, etc.—are dependent on the size and location of the tumor mass. The nasal obstruction is of a slow, progressive nature, usually accompanied by every evidence of acute or chronic infection of the respiratory tract. Associated with this symptom there is often a history of a sense of fulness or obstruction to one or both eustachian tubes. The latter is also often accompanied by tinnitus and progressive deafness of varying degrees. Another not infrequent symptom is postnasal hemorrhage, which occurs as a result of trauma to the friable soft tumor. This is seen more commonly with growths involving the nasopharyngeal vault. A sneezing spell or forceful blowing of the nose may precipitate a profuse hemorrhage. Needless to say, naso-

5. Cloward, R. E.: Nasopharyngeal Cancer, Arch. Otolaryng. **32**:512 (Sept.) 1940.

pharyngoscopy, posterior rhinoscopy and digital examination of the nasopharynx should be included in the usual routine examination. These procedures will undoubtedly reveal any minute or obscure pathologic process that may be present.

Often, owing to its slow, insiduous nature, the growth is undiagnosed until metastasis appears, which is most commonly to the cervical lymph chain. Later, metastasis may take place to the lung, the abdominal region, etc. Intracranial invasion is not uncommon, occurring by extension and direct erosion.

The prognosis depends first, on early diagnosis, and, second, on metastasis either by the lymph channels to the cervical and other lymph glands or by intracranial extension. Owing to the high radiosensitivity of this tumor, a reserved favorable prognosis can be given when the lesion is detected prior to any extensive metastasis. Even in some cases in which metastasis to a few cervical nodes has taken place, remarkable disappearance of the tumor may occur.

The treatment of choice is high voltage roentgen radiation, given intensively until 4,000 to 6,000 r has been administered. My patient received fourteen daily treatments of 200 r each, directed to alternate sides. As a rule one series is sufficient to bring about a complete cure, but in a few cases, especially those in which lymphadenopathy or intracranial extension exists, the series may have to be repeated.

SUMMARY

A case of lymphoepithelioma of the tonsil and nasopharynx is reported. The interesting features of this case lie, first, in the occurrence of the growth in the tonsil; second, in the belated appearance of cervical adenopathy (probably metastatic); third, in the appearance of the tumor in the nasopharynx, which would have been unrecognized if it had not been for the occurrence of the profuse hemorrhages; fourth, and finally, in the prompt regression of the growth after the institution of high voltage roentgen therapy.

CONCLUSIONS

1. Lymphoepithelioma may occur at two sites, either simultaneously or at different times.

2. Early diagnosis is of prime importance. Hence, nasopharyngoscopy and posterior rhinoscopy, together with digital examination, are essentials in every case of slow, progressive nasal obstruction, progressive deafness, either unilateral or bilateral, especially in the fourth, fifth or sixth decade of life, and spontaneous, unaccountable hemorrhage from the nose or the nasopharynx.

3. Biopsy should be made of all pathologic masses in this region.

4. Intensive high voltage roentgen irradiation is the only method of treatment, since this tumor is extremely radiosensitive. From 4,000 to 6,000 r should constitute the average dose in a continuous series of treatments.

5. While the prognosis is guarded, it is rather favorable in the early stages, particularly when metastasis is absent or, if present, is limited to only a few cervical lymph nodes.

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SEQUESTRATION OF THE OSSEOUS LABYRINTH

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Occasionally in the course of acute and chronic suppurations of the middle ear sequestrums develop as a result of necrosis of the osseous labyrinth. This complication may appear in the course of scarlet fever, cholesteatoma, tuberculosis, diabetes, syphilis and other types of granuloma and in the case of injury to the osseous labyrinth during surgical procedures on the ear. We believe, too, that petrositis is occasionally complicated by necrosis and sequestration of the bony labyrinth. The disease occurs most often in children, but it may appear at any age. The extent may vary from slight circumscribed caries to involvement of the entire temporal bone, and even of the surrounding occipital, zygomatic and parietal bones.

SYMPTOMS

Pain occurs frequently, and it is usually severe. It may be due to inflammation of the adjacent periosteum or meninges or to retention of the products of suppuration and frequently disappears only on removal of the sequestrum.

Early in the disease tinnitus, profound deafness, vertigo and vomiting may develop as a result of invasion of the inner ear. Eventually the cochlear and vestibular end organs become completely destroyed. The discharge from the ear is usually copious and thick, and granulations are exuberant.

Paresis or paralysis of the face is frequent. It is due to interstitial neuritis associated with necrosis of the fallopian canal.

PATHOLOGIC FEATURES

The otitic capsule is made up of three layers of bone. The inner, or endosteal, layer is thin and compact. The middle, or enchondral, layer is made up of primitive bone laid down by ossification of the cartilaginous capsule. It is characterized by remnants of calcified cartilage scattered throughout. Vascularization is extremely scant in this layer, and there is slight tendency to healing in the event of injury (fractures) or inflammatory destruction. The outer, or periosteal, layer contains haversian lamellas and is richly endowed with blood vessels. Destruction of this layer of bone is followed by prompt regeneration.

The great majority of inflammatory reactions occur in the air cell system and limit themselves to the periosteal layer. Should the enchondral layer become involved, the necrosis may result in development of a labyrinthine fistula or in the formation of sequestrums of enchondral bone.[1] If healing should occur, the

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1. Nager, F. R.: Pathology of the Labyrinthine Capsule and Its Clinical Significance, in Fowler, E. P., Jr.: Medicine of the Ear, New York, Thos. Nelson & Sons, 1939.

defects in the endosteal and enchondral layers will be replaced by compact lamellar bone derived from the periosteal layer.

Pneumatization in the pyramid occurs entirely in the periosteal layer. Because of the larger mass of periosteal bone which develops above and behind the labyrinth, medial to the arcuate eminence, there is a tendency to development of pneumatized areas in that location. Because of the narrow exit from these regions to the mastoid cells or the middle ear proper, there is a tendency to inadequate drainage during suppuration. Such a process predisposes to necrosis of the labyrinth, as well as to direct invasion of intracranial structures.

In cases of pyogenic suppuration the most common pathway of invasion of the labyrinth is by direct necrosis of the osseous capsule. Usually a semicircular canal is invaded as a result of suppuration in perilabyrinthine cells. The presence of other factors, either systemic, such as diabetes mellitus, or local, such as cholesteatoma, tuberculoma [2] and other granulomas, predisposes to invasion of the osseous capsule and the inner ear.

Once the infection gains the interior of the labyrinth it may be checked by the formation of granulations. This may occur locally near the site of the caries or, in the case of panotitis, in the porus acusticus internus and in the cochlear and vestibular aqueducts. If adequate drainage is established, healing may occur by fibrosis and ossification. Otherwise, necrosis and sequestration of the labyrinth may begin and either part or all of the labyrinth be walled off by granulations. Should the formation of granulations fail to prevent the infection from extending to the meninges, labyrinthine meningitis or cerebellar abscess results. The meninges may become infected by extension of the infection through the porus acusticus internus, through the cochlear aqueduct, through the vestibular aqueduct, with rupture of the saccus endolymphaticus [3] or by extension from any extradural focus of suppuration.

Empyema of the saccus endolymphaticus tends to produce a cerebellar abscess because of the formation of adhesions to the cerebellum, affording a pathway for direct infection.[4] A focus of suppuration in perilabyrinthine cells may, similarly, lead to invasion of either the middle or the posterior cranial fossa.

A sequestrum may vary in size from a small portion of the bony capsule to an entire labyrinth. The lesion may be single or multiple. It may migrate, usually toward the external canal. The progress of the disease may be arrested by the formation of a strong connective tissue barrier and by hyperplasia and osteosclerosis of the surrounding viable bone.

When the sequestrum is discharged or removed, the resultant cavity quickly fills up with granulations, which change into fibrous tissue, and gradually ossifies. This ossification may proceed and fill the tympanum, and even the external auditory canal. The cavity may, however, become lined with stratified squamous epithelium and form cholesteatomatous masses.[5]

DIAGNOSIS

The presence of a sequestrum is usually difficult to diagnose before surgical exposure. Roentgen studies will frequently suggest erosion or necrosis of the bony

2. Proctor, B., and Lindsay, J. R.: Tuberculosis of the Ear, Arch. Otolaryng. **35**:221-249 (Feb.) 1942.

3. Lindsay, J. R.: Suppuration in the Petrous Pyramid, Ann. Otol., Rhin. & Laryng. **47**:3-36, 1938.

4. Bezold, F., and Siebenmann, F.: Text-Book of Otology for Physicians and Students, Chicago, E. H. Colegrove Co., 1908.

5. Politzer, A.: A Text-Book of the Diseases of the Ear, London, Baillière, Tindall & Cox, 1909.

labyrinth and may in rare instances demonstrate a sequestrum. The sequestrum may be detected by probing, and in cases of persistent otalgia, exuberant granulations, profuse otorrhea and profound loss of function its presence should be suspected.

TREATMENT

Any suppurating ear with signs of impending invasion of the labyrinth demands immediate operation. When function is still present, the object of surgical intervention is to remove all foci of necrosis and suppuration without disturbing the labyrinth. This includes foci which may exist in pneumatized areas in the pyramid. A simple mastoidectomy permits exenteration of mastoid cells and drainage of perilabyrinthine areas. In cases of chronic suppuration and anterior petrositis the radical mastoid operation is preferable. It is also indicated when function has been destroyed. Care is necessary in removing sequestrums because of danger of injuring the meninges or the facial nerve. If a sequestrum is firmly attached it is advisable not to remove it forcibly but to wait until separation has become more complete.

PROGNOSIS

Once the sequestrum is removed and all foci in the temporal bone are adequately drained, the disease process stands a reasonably good chance of resolving. Foci remaining in the air cells of the petrosa may lie dormant for years and then suddenly flare up, producing necrosis of the osseous labyrinth.

A sequestrum may be expelled spontaneously and the ear heal, or it may be absorbed, with or without permanent deformity of the bony framework of the labyrinth. Progression of the disease process may produce death from meningitis, abscess of the brain, sinus thrombosis or erosion of the carotid artery or the lateral sinus.

In the event of paresis of the facial nerve, complete recovery may occur on subsidence of the infection. Complete paralysis is likely to result in some permanent loss of function. Destruction of the nerve trunk and disruption of continuity require repair by means of a nerve transplant or an anastomosis.

REPORT OF CASES

CASE 1.—W. E., aged 6 years, had scarlet fever in May 1941. He was desperately ill at the onset of the disease, with vomiting, high fever and dizziness. On the fourth day of the illness weakness of the right side of the face was noted, and a bilateral myringotomy was performed. A week later the patient was taken to a hospital, where bilateral simple mastoidectomy was carried out. Two weeks later he was discharged, with profuse otorrhea bilaterally and persistent paralysis of the right side of the face.

The left ear ceased to discharge in the next few months, but the right ear continued to drain postaurally, as well as through the external canal. Polypi began to appear in the external canal in August. Vestibular examination in September showed faint spontaneous nystagmus to the left. Caloric tests revealed absence of response on the right. The patient was unable to hear a loud shout into the right ear when the Bárány noise box was applied to the left ear.

Roentgenographic examination at this time revealed sequestration of the superior semicircular canal on the right (fig. 1 *A* and *B*). In the Stenver projection the superior perilabyrinthine area appeared to be pneumatized but clouded bilaterally.

Radical mastoidectomy, decompression of the facial nerve, removal of the sequestrum (fig. 2) and labyrinthectomy were performed on the right ear on Oct. 13, 1941. The middle ear and the external canal were filled with granulation tissue. Granulations were traced into the subarcuate fossa, and the sequestrum visualized in the roentgenogram was found and removed. The facial nerve was next exposed from its entrance into the middle ear down to the stylomastoid foramen. It was edematous and dark blue. The eminence of the horizontal semicircular canal was necrotic. It was carefully curetted, with exposure of the lumen of the canal, which was filled with granulation tissue.

The labyrinth was then opened superiorly, adjacent to the ampullary end of the superior semicircular canal. Another opening was made between the oval and the round window. Careful examination of the interior of the labyrinth revealed granulation tissue about the orifices of the semicircular canals. The remainder of the labyrinth was free of granulations, and the bony walls were smooth and shiny.

The patient had an uneventful convalescence. The ear was dry within a month after operation. The facial nerve has remained paralyzed almost completely up to the time of writing.

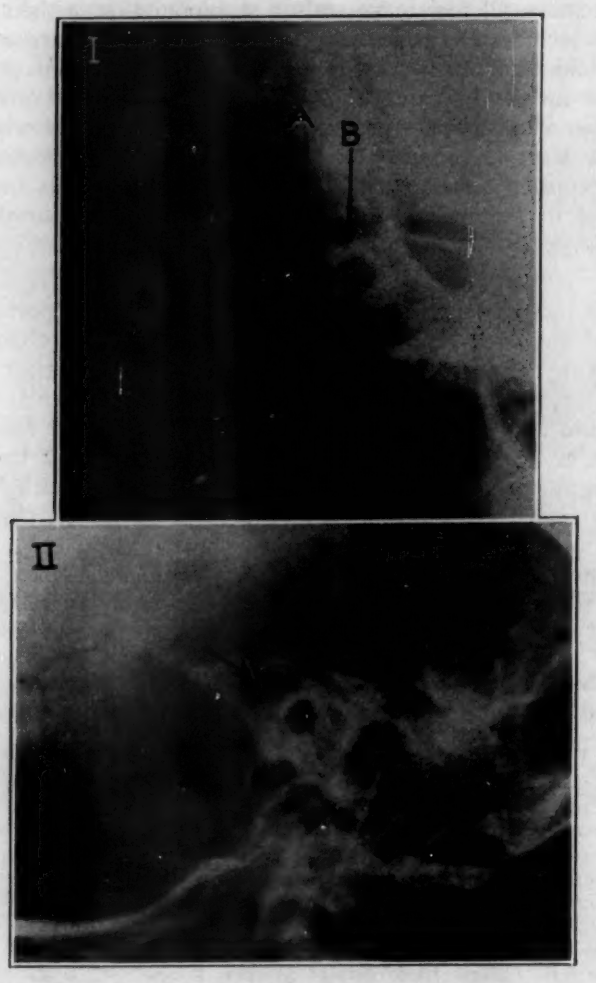

Fig. 1 (case 1).—*I*, Stenver's view of the right temporal bone. The sequestrated superior semicircular canal (*A*) is displaced laterally toward the external auditory canal. The superior perilabyrinthine area (*B*) shows evidence of necrosis.

II, lateral view of the right temporal bone. Note dehiscence between the crest of the superior semicircular canal and its descending posterior limb.

The symptoms at the onset of the aural disease in this case suggest early invasion of the labyrinth and of the facial nerve within the fallopian canal. The picture, then, is that of acute suppurative labyrinthitis which destroyed the vestibular and cochlear end organs. Persistence of the facial paralysis, profuse otorrhea and appearance of polypi in the ear are usually associated with the development of sequestrums.

It is uncommon to find roentgenologic evidence of sequestration, but a diagnosis of sequestration of the superior semicircular canal was made preoperatively in this case (fig. 1 *A* and *B*). Only 3 cases have been reported in which a preoperative diagnosis was made by means of roentgenograms.[6] In the case reported by Welin [6c] the condition was associated with petrositis of three years' duration.

Fig. 2 (case 1).—Photomicrograph of a section of the sequestrum containing the superior semicircular canal (*S.C.*). The lumens of the ascending and descending limbs are shown filled with granulation tissue. The edges of the sequestrum show irregular necrosis, with adherent organized granulation tissue.

6. (*a*) Kraus, L.: Felsenbeinsequestrationen im Röntgenbild, Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **28**:431-448, 1931. (*b*) Amersbach, K.: Ueber Sequestration im Bereich des Felsenbeins und die Bedeutung der Röntgendarstellung für deren Diagnose, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **133**:253-264, 1932. (*c*) Welin, S.: Case of Sequestration of Semicircular Canal Diagnosed by Radiography, Acta oto-laryng. **28**:181-186, 1940.

In the Stenver view (fig. 1*B*) it is seen that the sequestrum had migrated laterally 2 to 3 mm. toward the external auditory canal.

The appearance of the vestibule at operation, with granulations visible at the orifices of the superior and horizontal semicircular canals, but with the bulk of the vestibule free of granulations, suggests that the original labyrinthine infection had resolved but that development of caries of the walls of the superior and horizontal semicircular canals had produced a localized barrier of granulation tissue.

CASE 2.—L. G., aged 4 years, had scarlet fever in the spring of 1940. This illness was complicated by otitis media and mastoiditis on the left side, which necessitated a simple mastoidectomy at that time. The ear failed to respond to treatment and continued to discharge postaurally.

A year later, on May 15, 1941, the patient was admitted to the Children's Hospital of Michigan, and a secondary simple mastoidectomy was performed. Bony sequestrums were

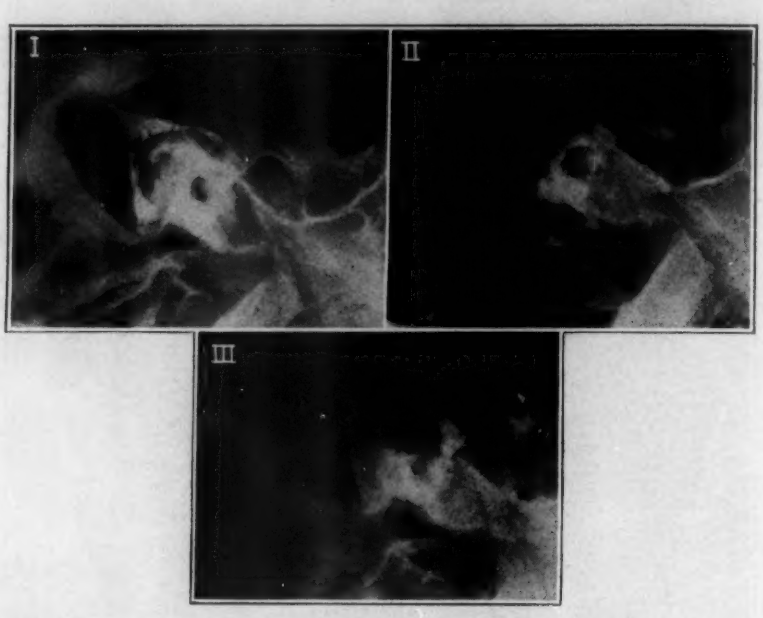

Fig. 3 (case 2).—*I*, lateral view of the right temporal bone on May 22, 1941. Sequestrums had been removed from between the sinodural angle and the labyrinth one week previously.

II, lateral view of the right temporal bone taken three months later (August 17), showing extensive necrosis of the superior semicircular canal.

III, another view of the same temporal bone taken on November 8, 1941, showing extensive destruction of the osseous labyrinth. The following day large masses of sequestrums were again removed from the temporal bone.

encountered between the sinodural angle and the labyrinth and removed. Immediately after the operation the child became febrile, and measles developed. The ear continued to discharge postaurally, and exuberant granulations formed in the postaural fistula. On June 23 complete paralysis of the left side of the face developed, after three days of otalgia and fever. On August 21 a radical mastoidectomy was performed. The middle ear contained large masses of granulations, which extended posteriorly behind the horizontal semicircular canal into the petrosa, where sequestrums were observed and removed. Examination of these sequestrums revealed portions of the bony semicircular canals.

After operation the ear continued to drain profusely, and granulations were exuberant. On November 7 the child began to complain of pain on the right side of the head. Within a

few hours she began to vomit and became drowsy. On November 8 nuchal rigidity was noted. The retinal vessels were congested, and the margin of the left disk was blurred. Faint spontaneous nystagmus to the right was present. The Kernig sign was elicited. Knee jerks were hyperactive. There were suggestive atonia and ataxia of the left hand. The spinal fluid was cloudy; the Pandy reaction was 2 plus; sugar measured 43.7 mg. and proteins 148 mg. per hundred cubic centimeters, and the white cell count was 1,790 per cubic millimeter, with 56 per cent polymorphonuclear leukocytes; smears revealed no organisms.

Examination of the blood revealed leukocytosis, with a count of 15,500.

The progressive destruction of the osseous labyrinth is illustrated in figure 3 *I, II* and *III*.

The left temporal bone was surgically explored on November 9. Large masses of granulation tissue were removed, together with numerous sequestrums. A probe was gently passed, without resistance, into the depth of the cavity in the direction of the internal auditory meatus. On removal of the probe blood-tinged spinal fluid escaped. The wound was filled with iodized poppyseed oil, and roentgenograms were made (fig. 4). On completion of the roentgenographic studies the wound was filled with sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole).

The child was immediately placed under treatment with sulfadiazine (2-[paraaminobenzenesulfonamido]-pyrimidine), 7.7 grains (0.5 Gm.) every four hours, and given a transfusion. Lumbar puncture, repeated the following day, showed a pressure of 150 mm. of water, 54.5

Fig. 4 (case 2).—Mentovertical view taken on November 9, showing iodized poppyseed oil in the cavity after removal of sequestrums on the right. The right apex appears sclerosed as compared with the left. The oil appears to replace the bony capsule almost completely.

mg. of sugar and 112 mg. of protein per hundred cubic centimeters, 405 white cells per cubic millimeter, with 40 per cent polymorphonuclear leukocytes, a negative Kahn reaction and a sterile culture. The number of white blood cells had dropped to 11,900 from a count of 15,500 on the previous day. On the second postoperative day the patient became stuporous; the left arm was flaccid; the retinal vessels were full; the reflexes of the left leg were hyperactive, and the Babinski sign was positive on the left side. Plans were made to explore the cerebellum for an abscess, but the patient died before operation was performed. Permission for autopsy was refused.

This case, again, is one of sequestration following scarlet fever. The course was more insidious than that in the first case. A history of acute labyrinthitis was not elicited, and the facial paralysis appeared late in the course of the disease. The operation on May 15 yielded sequestrums from the posterior portion of the labyrinth. Residual infection in a group of posterior and superior perilabyrinthine cells probably was responsible for the development of caries at the level of the posterior semicircular canal and invasion of the fallopian canal in the proximity of the ganglion.

The final episode was most likely due to a cerebellar abscess. The operation on November 9 was performed to remove all remaining sequestrums and to establish adequate drainage of the source of infection in the temporal bone. Unfortunately, the child died before surgical drainage of the cerebellar abscess could be instituted.

Fig. 5 (case 2).—Photomicrograph of a sequestrum removed at operation. One of the walls of a semicircular canal is intact. Note the necrosis of the endosteal bone, with the organized exudate extending into the cavity (*A*). The sequestrum is made up of endosteal and enchondral bone.

CASE 3.—R. B., a man aged 63, was first seen at the University of Chicago Clinics on Nov. 6, 1935, complaining of drooping of the right side of the face for two months and difficulty in vision for six months. He was known to have been diabetic for the past ten years. The onset of the present illness dated back nineteen months, when there had developed a head cold, severe pain in the right ear, spontaneous otorrhea and severe pains in the right

temporal region, behind the ear and along the mandible to the midline. The pain was sometimes present below the eye. Dizziness was severe at the onset but had decreased. Diplopia was noted for several months.

Examination revealed complete paralysis of the right side of the face. No response was elicited with the faradic current. The reaction to the galvanic current was positive. Intermittent alternating divergence of the eyes was present, but no paresis of the ocular muscles was noted.

Otológic investigation revealed a scarred and retracted drum membrane on the left side. A purulent discharge was present on the right, and there was sagging of the roof of the canal. Hearing for the audiometer and for a loud shout was not elicited with the Bárány noise box in the left ear. Injection of ice water in the right ear failed to elicit a caloric response. Ten turns to the right in twenty seconds in the Bárány chair test produced an after-nystagmus to the left for sixteen seconds. Ten turns to the left produced an after-nystagmus of three or four jerks to the right.

Roentgenograms of the temporal bone revealed sclerosis in the antral and periantral regions bilaterally, with absence of air-containing cells on the right side. A few air-containing cells were present on the left. There was no evidence of destruction of bone or of cholesteatoma.

A radical mastoidectomy was performed on the right. The antrum was small and filled with granulation tissue. The bone of the prominence of the horizontal canal was necrotic and could be picked away with a hook. The tegmen was eroded near the geniculate ganglion. The facial nerve was observed to be incorporated in granulation tissue from the ganglion to below the level of the horizontal canal. The nerve, exposed for 0.5 cm. in its descending portion, appeared normal. A sequestrum containing the lateral wall of the vestibule was removed from the promontory. The postoperative course was uneventful. During the next four months several small sequestrums were extruded.

The patient was last seen six and a half years later. There was a slight return of facial movements. No further sequestrums had been extruded. The radical mastoidectomy cavity was dry.

Hearing tests, performed on March 28, 1942, showed no hearing in the right ear for audiometer or loud voice when the left ear was masked with a Bárány noise box. The monochord was heard on the right by bone and air conduction for several frequencies with the Bárány noise box in the left ear, but hearing for this also was eliminated when the ear was masked with a special device.

Diabetes was undoubtedly the factor predisposing to necrosis of the labyrinth and sequestration in this case. The facial paralysis resulted in permanent partial loss of function. Usually the amount of spontaneous recovery in such cases is as good as or better than that obtainable by surgical repair.

Of interest is the failure of the Bárány noise apparatus to mask out the monochord, although the hearing was unquestionably destroyed as far as the cochlear end organs were concerned.

CASE 4.—A. B., a woman aged 30, was referred to the University of Chicago Clinics on May 29, 1940. Her left ear had discharged continuously for eighteen years. On May 5 she caught cold and two days later had a severe pain in the left ear. The earache increased, until on May 10 dizziness, nausea and vomiting developed. Three days later pain developed in the forehead and in the back of the neck, with relief of the earache. Later in the day the patient became semicomatose and was admitted to the Municipal Contagious Disease Hospital of Chicago.

Examination revealed nuchal rigidity and some blurring of the optic disks, especially on the left. The left ear was discharging. There were 4,000 cells per cubic millimeter of spinal fluid. Culture of the fluid yielded a gram-negative, motile rod, which was not identified. Sulfanilamide therapy was started immediately and given in large doses until May 29. The spinal fluid on May 18 contained 600 cells per cubic millimeter. Cultures of this fluid were sterile. A mastoidectomy was performed on May 22, with release of pus from the antrum. The temperature ranged from 101 to 104 F. between May 13 and May 29. After operation the patient had severe pain over the right eye, which was kept closed. Diplopia was not present. The right fundus showed progressive blurring of the margins of the disks, with development of hemorrhages. Cultures of the blood were sterile. In view of signs of a persisting focus of suppuration, the patient was transferred to the University of Chicago Clinics on May 29.

The patient was fully conscious on admission. There was a purulent discharge from the left ear and from the wound in the left mastoid. Ophthalmologic examination showed papilledema of 2 D., with flame-shaped hemorrhages over the right retina. Diplopia was present on lateral gaze to the right. The visual fields were normal. There was slight pain over the right eye.

Examination of the spinal fluid on admission revealed 225 cells per cubic millimeter, chiefly polymorphonuclear leukocytes; 15.1 mg. of total proteins and 125 mg. of chlorides per hundred cubic centimeters; a sterile culture; a normal Wassermann reaction and colloidal gold curve; an initial pressure of 400 mm. of water, and normal dynamic readings. Culture of the discharge from the mastoid revealed Bacillus proteus and diphtheroids. Examination of the blood showed 7,200 white cells, 3,850,000 red cells and 84 per cent hemoglobin. The urine was normal. The blood pressure was 114 systolic and 78 diastolic. The general physical examination was essentially without significance.

Examination of the right ear revealed that the drum was intact and hearing practically normal. On the left the external canal and postaural wound were filled with pus and granulation tissue. The tympanic membrane had been destroyed, leaving no recognizable landmarks. The fistula test revealed no abnormality. There was no hearing for the audiometer or a loud shout when the right ear was masked with the Bárány buzzer. Spontaneous nystagmus to the right was observed on forward gaze. The cold caloric test, with 5 cc. of

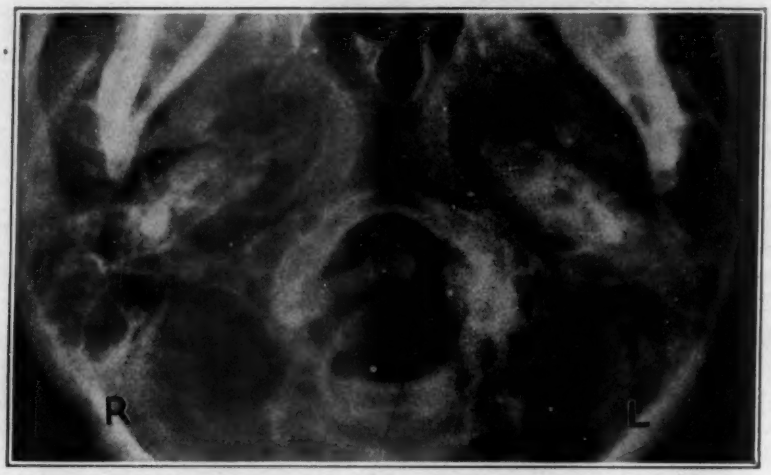

Fig. 6 (case 4).—Extensively pneumatized mastoid and petrosa on the healthy side (*R*); the apex appears irregular and more dense on the diseased side (*L*). The patient had had chronic suppuration since the age of 12 years.

water at 80 F., produced a normal response on the right. The same stimulus failed to elicit a response on the left. However, injection of 50 cc. of water at 40 F. in the left ear produced a definite increase in amplitude and frequency of the spontaneous nystagmus to the right for one minute and thirty seconds. Roentgenograms of the mastoids and the petrous pyramids (fig. 6) showed extensive pneumatization of the mastoid, perilabyrinthine region and apex on the right. The corresponding areas were irregular and more dense on the affected side.

The mastoid was surgically explored again on June 3. Large masses of granulations were removed, which revealed a large defect in the lateral sinus plate. A few remaining cells in the mastoid were completely exenterated. The group of cells between the antrum and the arcuate eminence were necrotic, and a sequestrum was removed from this area, which left the arcuate eminence intact and smooth. A radical operation was then performed. While granulations were being removed from the peritubal cell region a profuse hemorrhage occurred, which necessitated leaving a pack in position in the mouth of the tube.

The postoperative progress was fairly good. When removal of the packing from the mouth of the eustachian tube was attempted on the seventh postoperative day, a profuse hemorrhage again recurred, which necessitated repacking. This pack was finally removed three weeks after the radical operation. The patient complained of moderate intermittent pain in the ear for several months, and the aural discharge persisted. On Sept. 25, 1940 a

sequestrum was removed from the ear and sectioned (fig. 7). Partial paralysis of the face, which was noted shortly before the sequestrum came away, eventually disappeared. Granulations removed on November 12 for guinea pig inoculation proved to be negative for tuberculosis. On November 19 spontaneous nystagmus to the right was still present. It was aggravated by shaking the head. Caloric stimulation of the left ear with ice water (10 cc.) increased the spontaneous nystagmus in two tests and had no influence in two other tests. The caloric response on the right was normal. The rotation test with the patient in the Bárány chair produced an after-nystagmus for fifteen seconds after 10 turns to the right in twenty seconds. Ten turns to the left in twenty seconds produced an after-nystagmus of thirty seconds' duration. The left ear was totally dry by Aug. 12, 1941. Hearing tests performed on March 27, 1942 showed total loss of cochlear function on the left by both bone and air conduction when the hearing in the right ear was masked.

The history and observations in this case clearly indicate the sequence of events to have been: (1) chronic suppuration; (2) an acute cold and acute exacerbation

Fig. 7 (case 3).—Photomicrograph of a group of small sequestrums removed at operation. The bone is compact for the most part and is suggestive of enchondral bone in some areas.

of the otitis media; (3) diffuse suppurative labyrinthitis; (4) diffuse meningitis, with a cell count of 4,000 white cells per cubic millimeter, and papilledema, but with the type of organism unknown.

At the time of examination, on May 29, 1940, the meningitis had subsided to a large degree, but the cell count of the cerebrospinal fluid, the headaches and the persistent profuse aural discharge indicated that the focus of suppuration in the bone had not been adequately drained and was still producing meningeal irritation. Surgical exploration revealed perilabyrinthine necrosis with sequestration. It appears in this case that extensive pneumatization of the pyramid was an important factor predisposing to continued, deep-seated suppuration and to the development of complications during the acute exacerbation.

The increase in the amplitude of the spontaneous nystagmus on strong caloric stimulation at the time of admission is unexplained, since it is difficult to conceive

of residual vestibular function after suppurative labyrinthitis had run its course. Thermal stimulation of vestibular nerve fibers or Scarpa's ganglion may be a possibility.

SUMMARY

Four cases of sequestration of the osseous labyrinth are presented. In 2 cases the lesion occurred in children after scarlet fever. In these 2 cases the presence of suppuration in perilabyrinthine cell tracts predisposed to the complication. In the third case the condition was associated with diabetes. In the fourth case the complication was precipitated by an acute exacerbation of a chronic suppurative process in an ear with an extensively pneumatized mastoid and pyramid. These cases illustrate the importance of suppuration in pneumatized tracts deep in the pyramid in the pathogenesis of invasion of the labyrinth and sequestration and of intracranial complications. Occasionally suppuration in mastoid cells may result in necrosis of the bony capsule.

Erosion of a semicircular canal sometimes occurs as a complication of cholesteatoma or of granuloma. Attacks of vertigo result, and the result of the fistula test is usually positive. The caries may be localized or may be extensive enough to destroy the blood supply around a mass of intact bone with production of a sequestrum.

In the 4 cases presented there is no conclusive evidence of residual cochlear or vestibular function. It is possible to have residual cochlear, and even vestibular, function if only a portion of the osseous labyrinth has been sequestrated, but in the majority of cases early in the course of the disease suppurative labyrinthitis occurs which destroys the receptor organs.

Paralysis of the face was complete in 3 of the 4 cases. In the fourth case transitory facial paresis was observed in the course of the disease. The intimate anatomic relationship of the facial nerve to the osseous labyrinth is responsible for the frequency with which this nerve is involved.

In 2 of the 4 cases the picture was complicated by intracranial extension. In the second case a cerebellar abscess developed, and the patient died. In the fourth case meningitis developed, which responded to surgical intervention and chemotherapy.

The presence of sequestrums was discovered at operation in 3 of the 4 cases. In the fourth case (case 1) the diagnosis was made before operation by roentgenographic studies.

CONCLUSION

1. Suppuration in the perilabyrinthine cell groups (petrositis) plays an important role in the development of invasion of the inner ear, necrosis and sequestration of the osseous labyrinth.

2. Hearing and vestibular function are, as a rule, completely destroyed in this disease process.

3. Paralysis of the face is a common complication.

4. Treatment should be directed toward removal of the sequestrum and complete removal of all foci in the temporal bone. A radical mastoidectomy is usually necessary. An operation on the petrous pyramid may be required.

5. The existence of a sequestrum frequently becomes evident only at operation, but occasionally may be demonstrated by roentgenograms.

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CONTROL OF HEMORRHAGE

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During experimental research in blood coagulation Steinberg and Brown, of the Kensington Hospital for Women of Philadelphia, found that the intravenous or intramuscular injection of oxalic acid in experimental animals produced a rapid fall in the blood coagulation time of these animals. Thus, the results which they observed were diametrically opposed to those obtained in vitro. Oxalic acid is a chemical found in abundance in nature and has long been known to be toxic when administered in large doses. Solomon stated that the lowest recorded fatal dose was 5 Gm. and the lowest toxic dose 10 grains (0.65 Gm.), the latter being about the amount found in 1 pound (453 Gm.) of rhubarb greens. Oxalic acid is deemed to be rather inert in the body and appears to be excreted by the kidneys. Ingestion of large amounts of this substance appears to cause death by clogging the renal mechanism.

The blood of human beings has been found normally to maintain a constant oxalic acid level. By the use of a trimetric method of testing the oxalic acid content of the blood, normal values have been established at 5.5 to 7.5 mg. per hundred cubic centimeters of blood. By the same method blood values have been determined for conditions other than normal, and in many cases an increase in the blood coagulation time has been found to accompany a lowered oxalic acid concentration in the blood. Values for cord and maternal blood are found normally to be as high as 17 mg. per hundred cubic centimeters of blood. The protective value of this high content is obvious.

Inaccessible hemorrhage has long been enigmatic, both in medical and in surgical cases, and enumeration of the substances given orally or parenterally would indeed be not only an extensive one by an indication of the ineffectualness of any of them. Calcium in many forms, horse serum, moccasin venom, cephalin, vitamin K, salt pork directly applied, and many other substances have been empirically administered. Except with certain blood dyscrasies and vitamin deficiencies, the results obtained by the administration of these many substances have largely been unsuccessful. It is the patient whose blood is within the limits of normal who usually concerns the physician.

The experimental work carried out by Steinberg and Brown, and later by others, was with an extract of certain plants (shepherd's purse, wood sorrel, beets, oxalis, citrous fruits, alfalfa, etc.) the active prin-

ciple of which was found to be a chemical related to the dicarboxyl group. Since the chief component was found to be oxalic acid, it was felt that this substance could safely be used per se, inasmuch as chemically pure oxalic acid contains impurities which could in no manner be harmful when the original substance is given in the prescribed dose. Chemically pure oxalic acid was, therefore, made up in stock solution with distilled water, placed in stoppered bottles and autoclaved. One gram of oxalic acid was added to 1 liter of distilled water. The acid is freely soluble in this dilution and is apparently stable for some time. The injection of a few cubic centimeters of this solution into the human body is attended by a rapid and rather sustained fall in the coagulation time of the blood.

Any discussion of hemorrhage must obviously be complex if all factors are considered, and it is not the purpose of this paper to consider the various theories of blood coagulation and the vast number of important contributions to the subject of hemorrhage. For the purpose of this paper let it suffice to classify hemorrhage as mild, moderate and massive. The first type is of little consequence and is stopped by normally functioning blood elements. The second and third types are those which concern the otolaryngologist. Since blood coagulation at the site of origin of the hemorrhage depends on the fact that the elements in the blood responsible for coagulation must pause long enough to carry out their function, it follows that any substance placed in the blood stream must likewise pause long enough to reach the tissues locally. Without mechanical aid in transforming a massive hemorrhage into a moderate one this substance could not reasonably be expected to act.

In nasal and pharyngeal surgery the otolaryngologist not infrequently meets with operative, as well as postoperative, hemorrhage which stubbornly resists attempts to stop it. Severe hemorrhage of the nose, the origin of which is beyond the scope of inspection, is amenable to treatment with the solution of this acid indicated here, but usually requires supplementary packing to be brought completely under control. Although, fortunately, it is not a common experience, almost every one has seen the patient whose epistaxis continues despite postnasal and antenasal packing. I have seen several such patients whose hemorrhage has been stopped by the intravenous administration of a solution of oxalic acid.

The injection of the solution of oxalic acid raises the total level of the substance in the blood stream far above that which could be accounted for by the small amount administered. This phenomenon has not been explained. This material has not been found to be toxic when administered properly and in no way endangers the status of the patient. It appears to promote the production of prothrombin, as evi-

denced by the reduction of the prothrombin time after its use. It also increases blood viscosity and is antagonistic to heparin and hirudin. It appears from these facts that contraindications to its use are essential hypertension, peripheral vascular disease, thromboangiitis obliterans and allied conditions. If the hemorrhage in any given case is deemed more important than any of these conditions, its use should still be considered.

After extensive primary or secondary hemorrhage a patient may be a candidate for transfusion. This solution may then play a dual role in that it may be given to the donor, as well as the recipient.

CLINICAL APPLICATION

The explanation of the action of oxalic acid in the body is not yet clear, but the fact that it lowers considerably the coagulation time has been established with certainty. Since the action of this material is in the blood stream, it is obvious that the use of the solution need not be confined to the field of otolaryngology. Since the discovery of vitamin K many attempts have been made to use this vitamin with acute hemorrhage. The fact remains that its use is very limited in that it is of no value in cases of acute hemorrhage in which the liver and bile function normally.

Since this preparation lowers the coagulation time in normal persons, as well as those with certain abnormal conditions, it follows that it may be useful before as well as during and after operation. For the patient whose condition after operation would be endangered by any type of thrombosis this substance is obviously contraindicated. I have had considerable experience with the use of oxalic acid for patients with tuberculosis who experience hemorrhage. Examination of the blood expectorated by such patients who have received oxalic acid discloses an interesting and important fact. In almost every case, except those of a severe nature, the clotted blood has been noted to be firmer and less elastic than normal. It is reasonable to assume that a lesion surrounded by a clot of such consistency is more likely to remain localized than a lesion surrounded by one of less density. By the same line of reasoning, it would appear that an infected clot or thrombus theoretically can be assumed to be safer if its matrix consists of elements making it more firm and tenacious. Such assumptions are admittedly not without error, inasmuch as proving them by a logical coefficient is outside the realm of probability.

Any didactic attempt to regulate dosages in chemotherapy in a narrow sense is futile and misleading. The many factors at work in human tissues in which hemorrhage is encountered bar such an attempt. Certain basic doses may be expressed, however, without encroaching on this principle. The method of choice of administering the solution under

discussion is intravenous. Its use intramuscularly is attended by slight discomfort for a few minutes, but this should not bar the employment of this route if it is deemed the one indicated. As a prophylactic measure, 2 cc. may be given one to one-half hour prior to operation, followed by the same amount immediately afterward.

In cases of profuse hemorrhage 3 to 5 cc. may be given intravenously, followed in one-half hour by 2 cc. injected intramuscularly if the hemorrhage persists. Additional amounts of 2 cc. may be administered at hourly intervals if the hemorrhage does not stop, for 3 doses. Care should be taken to avoid the formation of a dense and tenacious clot in any of the air passages. Many of the distressing hemorrhages associated with the carotid blood vessels can be controlled or stopped by the judicious use of this solution, and its administration will, I feel, often prevent surgical mayhem on this system.

Once this solution has entered the blood stream, its action is almost immediate, and one adequate dose will persist in its activity for several hours. In the amounts recommended it has not proved to be toxic, in contrast to other substances administered for the control of hemorrhage. It contains no alkaloids or proteins, and side effects are not to be expected.

SUMMARY

The purpose of this paper is not to convey the impression that the use of oxalic acid is the complete solution to the ever present problem of hemorrhage, but to emphasize the fact that oxalic acid, in proper dilution and dosage, has proved itself to be a valuable agent in the control of moderate hemorrhage. It is economical, easily administered, nontoxic and efficient.

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SCHMINCKE TUMOR

LYMPHOEPITHELIOMA OF THE NASOPHARYNX

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We present a compilation of most of the literature on this supposedly rare tumor and report 3 cases of our own which we have encountered in the past year.

HISTORY

In 1921 Regaud and Reverchon [1] and Schmincke [2] published independently papers on this tumor. Reverchon and Cantard,[3] in a report from Regaud's clinic in 1921, originally noted a tumor consisting of two elements: a primitive type of epithelium constantly associated with lymphocytes. Schmincke, in the same year, reported several cases of this tumor of the nasopharynx and described the pathologic character.

In 1926 Jovin [4] discussed the histologic features of this tumor, describing it as a mass of epithelial cells with poorly delimited cytoplasm, forming at times a syncytium and being infiltrated with lymphocytes.

In the past few years a number of papers have appeared, most of which are controversial with regard to the actual classification of the tumor. However, they are all more or less in accord in their pathologic discussion of the neoplasm.[5]

DEFINITION

The Schmincke tumor is a malignant neoplasm of lymphoid tissue, usually in the pharyngeal region, characterized by a peculiar admixture of lymphoid cells intimately associated with epithelial elements of an immature squamous or epidermoid type, which tends to metastasize early and rapidly and is highly malignant and extremely radiosensitive.

CLINICAL MANIFESTATIONS [6]

The patient usually presents himself to the physician with a complaint of swelling in the neck. This is seen as a lymphadenopathy. However, a complete history will reveal other symptoms. In the order of frequency of their occurrence as found

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1. Regaud, C., and Reverchon, L.: Sur un cas d'epithélioma épidermoïde développé dans le massif maxillaire supérieur, étendu aux téguments de la face, aux cavités buccale, nasale et orbitaire, Rev. de laryng. **42**:369-378, 1921.

2. Schmincke, A.: Ueber lymphoepitheliale Geschwülste, Beitr. z. path. Anat. u. z. allg. Path. **58**:161-170, 1921.

3. Reverchon, L., and Cantard, H.: Lympho-epithéliome de l'hypopharynx traite par le roentgentherapie, Bull. et mém. Soc. franç. d'otol. et laryng. **34**:203-209, 1921.

4. Jovin, J.: Les lympho-epithéliomes du pharynx, Ann. d. mal. de l'orielle, du larynx **45**:729-758, 1926.

5. (*a*) Fitzhugh, W. M., Jr.: Lymphoepithelioma (Schmincke Tumor), Arch. Otolaryng. **28**:376-387 (Sept.) 1938. (*b*) Harvey, W. F.; Dawson, E. K., and Innes, J. R. M.: Debatable Tumours in Human and Animal Pathology: Lymphoepithelioma, Edinburgh M. J. **44**:549-556 (Aug.) 1937. (*c*) Martin, H. E., and Blady, J. V.: Cancer of the Nasopharynx, Arch. Otolaryng. **32**:692-727 (Oct.) 1940.

(Footnotes continued on next page)

through a study of the literature [7] and of our own cases, they are: (1) swelling of the neck; (2) pain or a sensation of fulness in the ear; (3) pain in the throat, the local pain often radiating to the neck and head; (4) nasal obstruction or spontaneous epistaxis; (5) double vision; (6) unilateral loss of vision; (7) pain or numbness of the face on one side; (8) visual paralysis; (9) ptosis, and (10) occasional dysphagia.

These symptoms may not all be present, for they develop only as a result of progression in the growth of the tumor, so that a diagnosis must be made from the early signs and symptoms. In fact, it cannot be too strongly emphasized that cervical adenopathy in the adult is frequently malignant, and when malignant it is usually of metastatic origin and should lead one to search carefully for a primary lesion in the upper respiratory tract.[5c] Examination of the patient may reveal, besides the palpable nodes in the neck, discoloration of the tympanic membrane, fluid in or a discharge from the ear, paralysis (unilateral or bilateral) of the abducens nerve and various signs indicative of involvement of other cranial nerves.

Roentgenographic examination sometimes shows destruction of the base of the skull and involvement of the sphenoid sinus, and possibly of the mastoid and petrous portions of the temporal bone. Examination of the nasopharynx often reveals a small tumor in the upper part of the nasopharynx and around the eustachian orifice.

DIAGNOSIS

Although one may postulate from clinical experience the diagnosis of the tumor to be visualized, a positive diagnosis of this condition can be made only from the microscopic section. One must differentiate the lesion from nonmalignant tumor, syphilis, tuberculosis, Hodgkin's disease, chronic infection, sarcoma and other types or carcinoma.[8]

We should like to offer a suggestion in the taking of nasopharyngeal tissue for biopsy. Martin and Blady [5e] said:

> In our experience, in no other part of the upper respiratory and alimentary tract is cancer so apt to be overgrown by hypertrophic lymphoid or granulation tissue as in the nasopharynx.

6. (*a*) Furstenberg, A. C.: Malignant Neoplasms of the Nasopharynx, Surg., Gynec. & Obst. **66**:400-404 (Feb., no. 2 A) 1938. (*b*) Black, J. I. M.: Clinical Manifestations of Cancer of the Nasopharynx, Newcastle M. J. **18**:112-122 (July) 1938. (*c*) Weinberger, N. S.: Cancer of the Nasopharynx, Ann. Otol., Rhin. & Laryng. **48**:203-211 (March) 1939.

7. (*a*) Christianson, O., and McArthur, S. W.: Nasopharyngeal Carcinoma, Arch. Surg. **27**:1109-1119 (Dec.) 1933. (*b*) Skorpil, F.: Ueber die Lymphoepitheliome (Schmincke) der Speicheldrüsen, Frankfurt. Ztschr. f. Path. **53**:450-466, 1939. (*c*) Boyd, L. J., and Goldbloom, A. A.: Schmincke's Tumor (Lymphoepithelioma), with Metastasis Complicated by Tracheo-Esophageal Fistula, New York M. Coll. & Flower Hosp. Bull. **1**:39-45 (April) 1938. (*d*) Burnam, C. F.: Diagnosis and Treatment of Malignant Tonsil Conditions, Surg., Gynec. & Obst. **55**:633-639 (Nov.) 1932. (*e*) Hauser, I. J., and Brownell, D. H.: Malignant Neoplasms of the Nasopharynx, J. A. M. A. **111**:2467-2473 (Dec. 31) 1938; (*f*) Malignant Neoplasms of the Nasopharynx, Tr. Sect. Laryng., Otol. & Rhin., A. M. A., 1938, pp. 278-296. (*g*) Furstenberg.[6a] (*h*) Martin and Blady.[5e]

8. (*a*) Black, J. I. M.: The Lympho-Epithelioma, J. Laryng. & Otol. **53**:225-245 (April) 1938. (*b*) Ewing, J.: Lympho-Epithelioma, Am. J. Path. **5**:99-107 (March) 1929; (*c*) Radio-Sensitive Epidermoid Carcinomas, Am. J. Roentgenol. **21**:313-321 (April) 1929; (*d*) Neoplastic Diseases, ed. 3, Philadelphia, W. B. Saunders Company, 1928. (*e*) Berven, E. G. E.: Malignant Tumors of the Tonsil, Acta radiol., 1931, supp. 11, pp. 1-285. (*f*) Boyd, W.: Textbook of Surgical Pathology, ed. 4, Philadelphia, W. B. Saunders Company, 1938. (*g*) Kienböck, R., and Selka, A.: Ueber das lymphoepithelioma der Rachenmandel (Schmincke's Tumor), Fortschr. a. d. Geb. d. Röntgenstrahlen **52**:227-233 (Sept.) 1935. (*h*) Berger, M.: Schmincke Tumors, New York State J. Med. **38**:193-195 (Feb. 1) 1938. (*i*) Dunlap, A. M.: Malignancy of the Nasopharynx and Eustachian Tube, Chinese M. J. **53**:68-71 (Jan.) 1938. (*j*) Goormaghtigh, N.: Cervical Reticuloendotheliomas: Differentiation from Lymphoepithelioma, Rev. belge sc. méd. **10**:147-158 (March) 1938. (*k*) Burnam.[7d] (*l*) Hauser and Brownell (footnotes 7 *e* and *f*). (*m*) Furstenberg.[6a]

As a result, the usual method of taking a specimen for biopsy with a straight biopsy forceps through the nose or with a curved one through the mouth has often proved unsuccessful and has necessitated several repetitions. In our experience, the use of an ordinary LaForce adenotome, as in adenoidectomy, especially after the area is visualized with a Blakesley soft palate retractor, has proved simple and effective in obtaining a large piece for biopsy.

ETIOLOGIC FACTORS

Age and Sex.—Middle or old age is the most common period for the occurrence of this tumor, but it has occasionally been seen at a much earlier age. The tumor has been more frequently observed in males.[9]

Incidence.—Ewing [10] observed the tumor in 4 per cent of malignant growths of the tonsil; Berven [8e] noted it in 6 per cent of such growths and in 11 per cent of malignant neoplasms of the nasopharynx.

Cause.—As with other neoplasms, the actual cause of the formation of the tumor is unknown.

PATHOLOGIC FEATURES

The name lymphoepithelioma has probably been derived from the fact that the tumor appears to arise from organs in which there is abundant lymphocytic infiltration of the submucosa.[5a] The primary growth is, therefore, not necessarily limited to the tonsil or the nasopharynx, but may be encountered wherever there are collections of lymphoid tissue. Proof of this is seen in the reported cases of involvement of the thymus, which is embryonically derived from the same group of tissues.[5a]

The primary lesion arises as a rule in Waldeyer's ring and is a soft, medium-sized, fungating neoplasm in the nasopharynx, the tonsil or the base of the tongue, which displaces rather than infiltrates the surrounding structures. It bleeds a little when touched, but is not painful. It has been seen in the parotid and in the submaxillary gland and occasionally in the tonsil.

According to Ewing [8b] and Boyd,[8f] this tumor begins and spreads in the structures concerned, which are intimately associated with the lymphatic system, appearing only on the surface and ulcerating later in the course of the disease. This accounts to a large extent for the early and constant metastasis to the lymph nodes. The affected lymph glands are usually bulky, and the process involves the upper deep cervical lymph nodes, generally bilaterally, with one side predominating. The mass is firm, but not hard. Deep fixation and lack of definite outline are common, but ulceration and involvement of the overlying skin rarely, if ever, occur. Distant metastasis is a terminal feature.[8g]

MICROSCOPIC FEATURES

In this tumor, carcinoma with immature cells, definitely of the primitive type, and absence of "pearl" formation and cornification has been observed.[5b] The separating tissue is essentially lymphoid in character, with an argyrophilic scaffolding. This separating tissue is in close relation to the epithelial elements and lymphocytes and may penetrate the mosaic of epithelial cells. The tumor itself is highly cellular and is composed of large, well defined cells with abundant granular cytoplasm [8g] and a vesicular nucleus. The nucleus is large, pale but well defined, with

9. Burnam.[7d] Hauser and Brownell (footnotes 7 *e* and *f*). Furstenberg.[6a] Black.[8a]
10. Ewing (footnotes 8 *b*, *c* and *d*).

one or more nucleoli.[5b] Mitosis is usually present.[8h] The proliferating tumor cells form solid alveolar bands, strands and masses. The cells are not everywhere sharply delimited from the stroma, and they resemble often a syncytium, the penetration of the mosaic of tumor cells by lymphocytes being one of its chief features.[11]

RELATION OF PATHOLOGIC STRUCTURE TO SYMPTOMS AND SIGNS

The interest in this tumor of the nasopharynx is not in its pathologic character but in its anatomic position, which gives rise to an interesting symptom complex.[8a] The primary growth is in the lateral part of the nasopharynx, often around the eustachian tube,[8i] and the tumor in growing spreads up through the foramens in the base of the skull and down through the lymph channels, and thus into the cervical nodes. Thus, this nasopharyngeal tumor and its extensions may be in close relation to the eustachian tube, the second, third and fourth nerves, the second and third divisions of the fifth nerve, the sixth nerve, the gasserian ganglion, the sella turcica, the jugular foramen and the ninth, tenth, eleventh and twelfth nerves.[12] It is the involvement of these structures which produces recognizable signs and symptoms of the disease as the tumor grows upward through the base of the skull and compresses the dura, rather than invades it. Any cranial nerve may be involved, but the seventh and eighth nerves rarely are so affected.[13] Because of the anatomic relations and the insensitivity of the parts, local symptoms do not appear early.

The rather extensive lymphoid apparatus draining this area and the smallness of the primary tumor, with its tendency to slow lateral spread, readily explain why the cervical adenopathy (the cervical chain of nodes being the direct drainage pathway of this area) is the earliest sign of this condition. As the primary tumor grows it obstructs the back of the nasal cavity and thus gives rise to the symptom of stuffiness of the nose. The soft, fungating character of the tumor, with its tendency to bleed easily, accounts for the impaired hearing and the pain in the ear, as well for the pain radiating to that area which is sometimes present. The signs of intracranial damage are due to extension of the growth, which tends to remain extradural; the dura forms the internal periosteum of the skull, and the growth as it advances through the foramen lacerum, which lies just in the roof of the nasopharynx, will lie extradurally, and therefore supraperiosteally, and strip the dura away from the internal table of the skull, which is therefore deprived of the protection of its resistant periosteum. For this reason, once the disease has extended through the foramen lacerum, the bone surrounding the foramen and the floor of the middle fossa is attacked from without and within, and erosion of the bone (if it occurs) is the result, rather than the cause, of the intracranial extension.[14]

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11. (*a*) Schlivek, K.: Schmincke's Tumor, Arch. Ophth. **7**:928-930 (June) 1932; (*b*) Ocular Manifestations of Schmincke Tumor, ibid. **17**:1055-1072 (June) 1937. (*c*) Needles, W.: Schmincke Tumor, J. Mt. Sinai Hosp. **1**:266-268 (March-April) 1935; (*d*) Cancer of the Nasopharynx with Special Reference to Neurological Complications: Clinical Study of Thirty-Five Cases, J. Nerv. & Ment. Dis. **86**:373-398 (Oct.) 1935. (*e*) Hirayama, J.: Lympho-epithelial Tumor (Schmincke Type) of Nasopharyngeal Vault, Gann **31**:363-369 (Aug.) 1937. (*f*) Cappell, D. F.: Pathology of Nasopharynx (Especially of Lymphoepithelioma), J. Laryng. & Otol. **53**:558-580 (Sept.) 1938. (*g*) Footnotes 6, 7 *a* to *f* and 8 *a* to *j*. (*h*) Harvey and others.[5b] (*i*) Martin and Blady.[5c]

12. Schlivek (footnote 11 *a* and *b*).

13. Needles (footnote 11 *c* and *d*).

14. (*a*) Capps, F. C. W.: Analysis of Some Tumors of the Nasopharynx, Nose and Maxilla, with Particular Reference to Occurrence of Lympho-Epithelioma, St. Barth. Hosp. Rep. **70**:50-77, 1937; (*b*) Lympho-Epithelioma in Nasopharynx, J. Laryng. & Otol. **54**:133-140 (March) 1939. (*c*) Druss, J. G.: Carcinoma of the Nasopharynx with Extension to the Petrous Pyramid, Laryngoscope **50**:359-365 (April) 1940. (*d*) Hirayama.[11e] (*e*) Black.[8a] (*f*) Martin and Blady.[5c]

The sixth nerve runs over the foramen lacerum and therefore is usually the first involved, with production of paralysis of the structures supplied. The third, fourth and fifth nerves lie close by and therefore are the next involved, causing signs referable to the ocular muscles, as well as neuralgia and pareses of the trigeminal nerve. When the disease extends forward, and thus into the orbit, it exerts pressure on the second nerve, with subsequent optic nerve atrophy. The seventh to the twelfth nerves lie posteriorly and are all involved later in the process of intracranial extension. Intraorbital extension occurs by way of the foramen lacerum, the carotid groove and the superior orbital fissure.[15]

TREATMENT

Treatment must take into account the primary lesion and the secondary metastases. For all practical purposes, the primary lesion in the nasopharynx must be considered anatomically unsuited to adequate surgical approach.[5c] The nasopharynx is comparatively inaccessible to surgical exposure. The area is essentially vascular, and growths of whatever size are poorly delimited, so that the difficulty of surgical removal, even of the small and more accessible lesions, is multiplied. Further, in cases in which surgical intervention has been attempted, recurrence has been frequent and rapid, and the recurring tissue has often exhibited a greater degree of malignancy. Since this tumor is very radiosensitive, irradiation is often the best hope of cure. There are now four methods in use: roentgen irradiation; interstitial application of radium; surface application of radium, and use of teleradium.

To quote from Burnam:[7d]

> The cure of a malignant condition by radiation fundamentally is based on the conception that malignant tumors can be destroyed by doses of rays which will not destroy, or seriously injure, normal surrounding tissues and structures. Both normal structures and malignant tumors vary greatly in their capacity to tolerate radiation. Some growths can be destroyed by radiation in quantities only about 1/20 of the amount known to be necessary to produce an erythema of the normal skin. On the other end of the scale, there are growths which will tolerate from 4 to 5 erythema skin doses. The essential reason for those variations is not known. It has been determined, however, that the microscopic architecture has an important bearing in estimating the probable effects of radiation. Tumors approaching adult, normal tissues are, for the most part, ray resistant. Tumors which are composed of undifferentiated embryonic-like cells are ray sensitive. . . . The tonsil is surrounded by normal tissues which, of themselves, are very ray sensitive and the injury of which may lead to pain, disturbance of nutrition, and many disagreeable symptoms. The mucous membranes of the mouth are more ray sensitive than the skin. The salivary glands are injured easily. There are many important nerves and blood vessels in this region. The periosteum of the hard palate and, more particularly, the mandible, are very ray sensitive. It is possible, by too heavy cross firing, to destroy easily large parts of the mandible. It is this fact which has made radiation of epithelioma of the floor of the mouth and gums so difficult. The results of treatment, as with malignancy everywhere, are dependent on the extent of the involvement. Immensely extensive neck and throat infiltrations may make it utterly impossible to carry out an effectual ray dosage throughout a tumor. Ray therapy has its technical limitations as definitely as surgery.

A good description of the radiation therapy of this condition may be found in the article by Martin and Blady.[8c]

PROGNOSIS

The condition, if untreated, is fatal in two or three years, the end being reached with massive local spread of the tumor. When treatment has been carried out and

15. (*a*) Bach, A. C.; Lederer, F. D., and Palevsky, S. N.: Extension Intracranially and Metastasis by Implantation into the Soft Palate: Clinical and Pathologic Study, Arch. Otolaryng. **31**:529-539 (March) 1940. (*b*) Footnote 11 *b* to *f*. (*c*) Footnote 14 *a* to *c*.

the diagnosis made very early, five year cures have been effected in many instances; with later treatment death often occurs from distant metastases.[8a]

The characteristic response to treatment is early and permanent regression of the primary and adjacent lymph nodes and of the possible distant metastases. If the local primary lesion responds satisfactorily to initial irradiation and regresses, it is unlikely that it will recur. If local regression is not obtained, death will soon follow. The percentage of cures have ranged from zero to about 25 per cent for five year cures, although Berven [8e] reported a series of cases showing five year cures in 50 to 75 per cent.

REPORT OF CASES

CASE 1.—S. D., an Italian shoemaker aged 56, was admitted to the hospital on Aug. 27, 1940 with the chief complaint of enlarged cervical nodes for about three months.

The past and family histories were irrelevant.

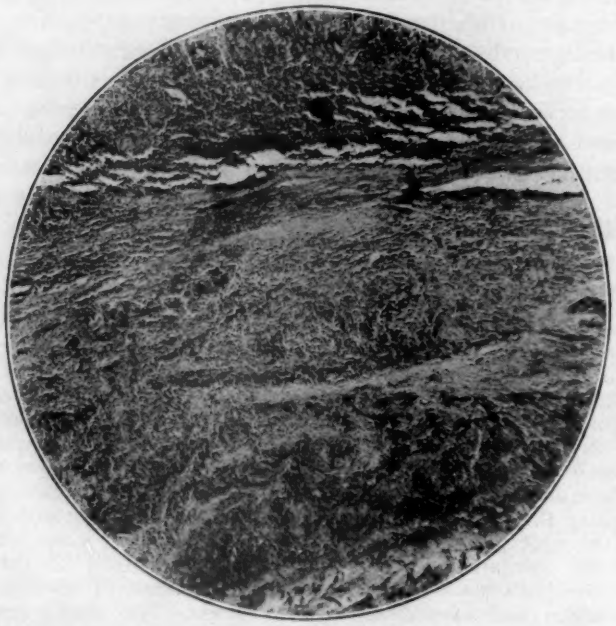

Fig. 1.—Tissue removed from nasopharynx, showing a syncytial mass of epithelial and lymphoid cells. Low power magnification.

Present Illness.—There was a history of gradually enlarging cervical nodes for about three months. No other swellings were noted. He had lost 5 pounds (2.3 Kg.) in weight. There was no history of weakness, fatigue or poor appetite. Occasionally he had had a bloody discharge from the nostrils after sneezing. On direct questioning he admitted a stuffy sensation in the nose.

Physical Examination.—The eyes were normal. There were old blood crusts in the nose. The teeth, gums, mouth and pharynx were normal, as were the heart, lungs, chest, abdomen, genitals and extremities. The reflexes were normal.

Glands: Two large, hard, discrete cervical nodes were noted on the right side; they were not attached to the skin and were freely movable. A small cervical node was noted on the left. The axillary nodes were moderately enlarged and freely movable. An enlarged submandibular node, removed for microscopic study, revealed carcinoma, of undetermined origin. Examination of the nasopharynx was advised. Consultation with an otolaryngologist was requested.

Otolaryngologic Examination.—There was respiratory difficulty with the mouth closed; with the mouth open respirations were free. Examination of the larynx disclosed no apparent obstruction to air. Inspection of the pharynx with the indirect mirror method revealed a pinkish red mass of irregular tissue which projected from the roof of the nasopharynx, spreading laterally from the midline. This mass bled profusely, even on gentle palpation. A biopsy specimen was taken with an adenotome; there was moderate bleeding, which was easily stopped; a slight ooze occurred for several hours. Microscopic examination revealed lymphoepithelioma, or Schmincke tumor (figs. 1 and 2).

The patient was discharged to Bellevue Hospital for radiation therapy. A follow-up report from that institution follows: "On Feb. 24, 1941, roentgenographic examination revealed moderate hypertrophic osteoarthritis involving the lumbar portion of the spine and both sacroiliac joints, as well as both hips. From February 14 to March 10 roentgen therapy (1,250 r) was given to the dorsolumbar area. The factors were: 200 kilovolts and 20 milliamperes; filtration, 0.5 mm. of copper and 1 mm. of aluminum; skin target distance, 50 cm., and size of portal, 10 by 15 cm., 150 r being delivered at each sitting. The patient was transferred to the psychopathic ward in March 1941."

He died April 11, 1941.

Fig. 2.—Syncytium of lymphoid and epithelial cells. The delimitation of cells from stroma is not everywhere sharp. The cells are large and well defined, with abundant granular cytoplasm and a vesicular nucleus. High power magnification.

CASE 2.—P. A., an Italian aged 86, was admitted on Nov. 14, 1940 with a history of soreness of the right side of the throat for several months. The right tonsillar area was ulcerated. A biopsy specimen (figs. 3 and 4) revealed lymphoepithelioma, or Schmincke tumor. The patient was transferred to Bellevue Hospital for radiation therapy. The report from that hospital follows: From November 28 to December 27 roentgen therapy was given to the right side of the neck, the total dose being 6,000 r. The factors used were: 200 kilovolts and 20 milliamperes; filtration, 0.5 mm. of copper and 1 mm. of aluminum; skin target distance, 50 cm., and size of portal, 8 by 10 cm., 250 r being delivered at each sitting. The patient was last seen on Jan. 14, 1941, at which time the primary tumor was destroyed. In March we received a telephone call from a relative of the patient, who stated that he was in the Metropolitan Hospital. We learned from this hospital that the patient died there April 5, 1941."

Autopsy.—The diagnosis was: "carcinoma of the tonsil, with extension to the tongue; purulent bronchopneumonia, and sepsis after operation for a Witzel fistula."

Fig. 3.—Carcinomatous tissue penetrating a syncytium of epithelial and lymphoid cells. Low power magnification.

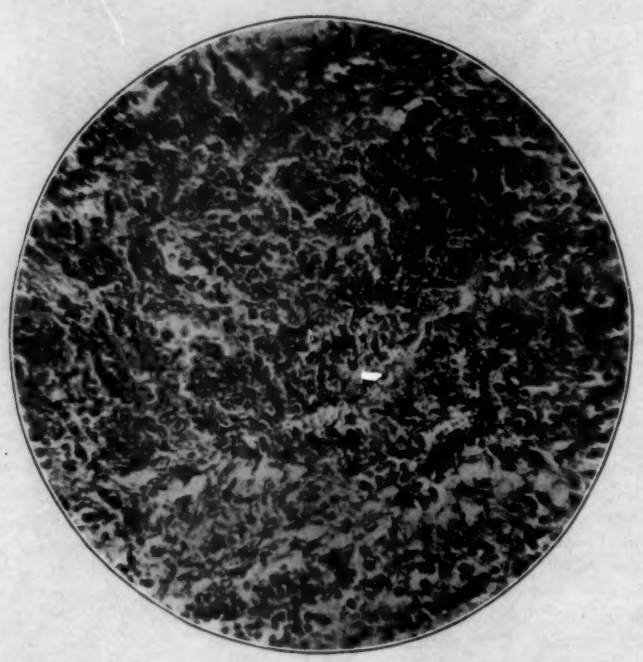

Fig. 4.—Biopsy tissue from right tonsil, showing carcinoma with immature cells, of the primitive type, without pearl formation or cornification. The separating tissue is essentially lymphoid and is in close relation to the epithelial elements and lymphocytes. High power magnification.

Fig. 5.—Section from the right tonsil, showing carcinomatous tissue of the primitive type with a separating tissue essentially lymphoid in character. The separating tissue penetrates the mosaic of epithelial cells. Low power magnification.

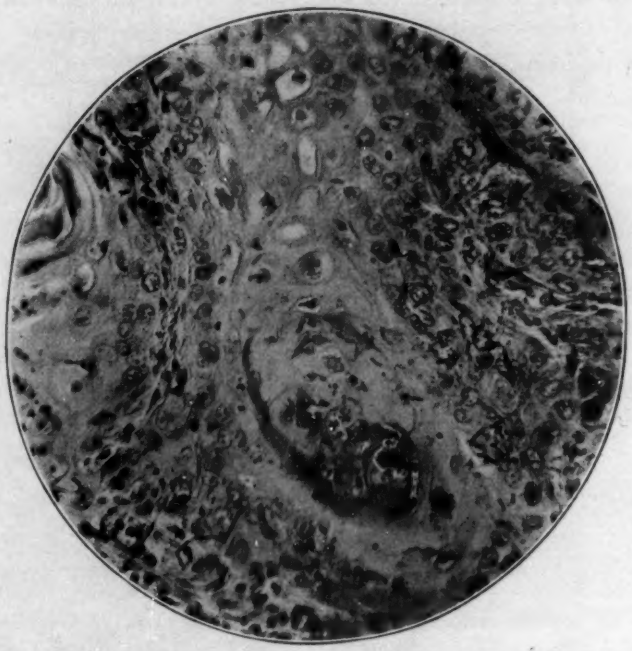

Fig. 6.—Tumor cells, showing a large, pale, well defined nucleus with one or more nucleoli. Mitosis is also seen in some of the cells. The proliferating tumor cells show solid alveolar bands, strands and masses. High power magnification.

CASE 3.—W. M., a white woman aged 62, was admitted on Feb. 5, 1941 with a history of progressive sore throat for seven months. For the last four months there has been swelling of the right side of the jaw and the submaxillary region. The patient had had an episode of "hemoptysis" on the day before, for which she sought admission to the hospital.

Examination revealed a swollen area in the region of the right tonsil, which bled easily on being touched. There was a foul odor from the mouth. The surrounding tissues were red and swollen. The right submaxillary region and the right anterior cervical nodes were swollen and very hard (figs. 5 and 6). A biopsy specimen was reported as showing squamous cell carcinoma of the tonsil (Schmincke tumor). The patient was sent to the Morrisania City Hospital for radiation therapy. From February 24 to March 11 she received 2,100 r of roentgen radiation in divided doses over the right tonsil and the upper portion of the neck and 200 r over the lower part of the face and the upper portion of the neck. She failed to return to the clinic after termination of the treatment.

COMMENT

Schmincke's tumor is frequently reported as a rare neoplasm. In the six month period from September 1940 to February 1941, 3 cases were encountered and the diagnosis verified by biopsy. From our own experience and a study of the literature we feel that this condition might be diagnosed more frequently and earlier, with a better prognosis afforded, if one were more alert and the nasopharynx were examined routinely.

SUMMARY

1. The history, clinical manifestations, pathologic features and treatment of Schmincke's tumor are discussed.

2. The pathologic picture in relation to the symptoms and signs is considered.

3. Three cases encountered in a period of six months, with photomicrographs, are reported.

4. A bibliography of most of the available literature on Schmincke's tumor is presented.

1235 Grand Concourse.

SURGERY OF THE MASTOID

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TORONTO, CANADA

Since the epoch-making contribution to otology by Ballance and Duel of the use of an autoplastic nerve graft for the repair of paralysis of the facial nerve, I have pursued this work from the experimental and the practical point of view. I have never been satisfied with the hit and miss method of simply placing a

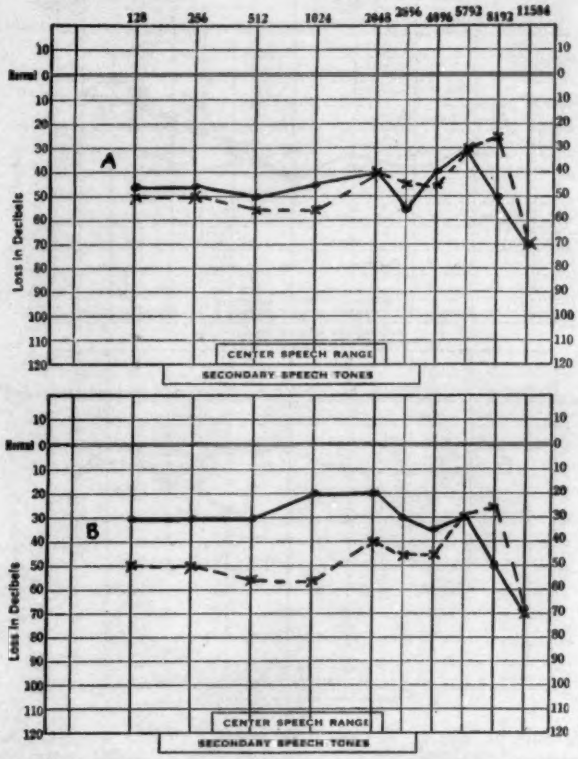

Chart 1.—Audiograms of Mr. B., aged 42, taken (*A*) one day before and (*B*) seven months after the operation. The solid line in these, and in the accompanying audiograms indicates the hearing loss, expressed in decibels, in the ear operated on, and the dotted line, the hearing loss in the intact ear. The fenestra nov-ovalis technic was used.

nerve graft in close approximation to the freshened ends of a divided nerve and hoping thus for a "maximum" degree of recovery.

In the discussion on the paper by McCaskey,[1] of Indianapolis, I stated:

Read before the Section of Otolaryngology, the Canadian Medical Association, Jasper, Alberta, Canada, June 19, 1942.

From the clinical services of otolaryngology, St. Michael's Hospital, and the Department of Otolaryngology, the University of Toronto.

1. McCaskey, C. H.: The Etiology and Treatment of Seventh-Nerve Paralysis, Tr. Am. Laryng., Rhin. & Otol. Soc. **46**:285, 1940.

In regard to the repair of the nerve, to ensure a satisfactory result, and to attain the maximum recovery it is essential to observe in nerve grafting the following two facts which are paramount; firstly, meticulous care and accuracy of technique in suturing in a non-infected field; and secondly, employment of a graft of the same calibre as the nerve, so that accurate approximation of the cut end of the graft and nerve can be obtained. I do not consider it sound surgery to repair a damaged nerve with a graft in an infected field.

Experimental work on nerve grafting, corroborated by clinical results, has conclusively proved that the downgrowth of neurofibrils is hindered by the presence of sutures, with the production of endoneural scar. I discussed this fact with Bentley,[2] of Manchester University, who has been working on the possibilities of peripheral nerve grafting.

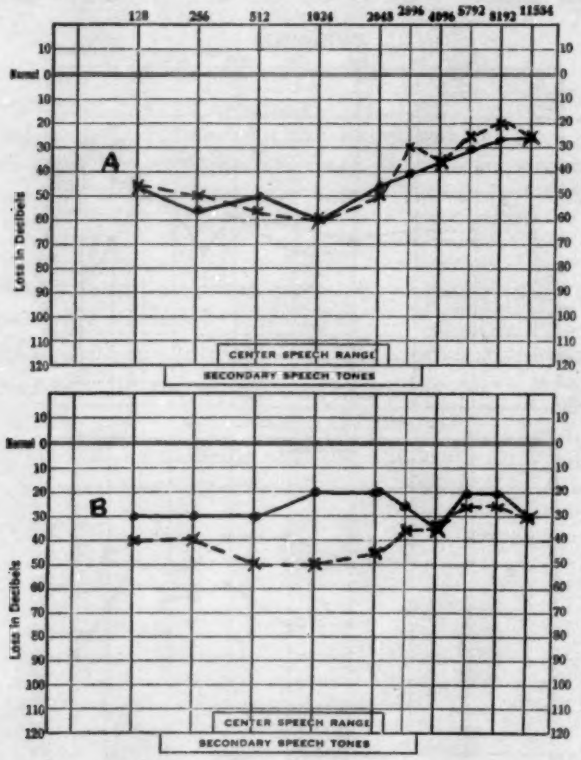

Chart 2.—Audiograms of Mr. G. A., aged 35, taken (*A*) one week before and (*B*) sixteen months after fenestration. The incus was removed. The patient is now married and has regained his position as a salesman.

To reduce the difficulties of nerve suture and to minimize the disorganization of the fibers which is apt to be produced by stitches, even when they are restricted as far as possible to the epineurium, a method has been devised by which the stumps can be held together with concentrated coagulated blood plasma. The method, introduced by Young and Medawar,[3] consists simply in holding the cut stumps together and pouring around them plasma which has just been mixed with a little strong tissue extract. For about 0.5 to 2 mm., depending on the age of the plasma and the strength of the extract, the plasma clots to a firm jelly, which

2. Bentley, F. H.: Personal communication to the author.
3. Young, J. Z., and Medawar, P. B.: Fibrin Suture of Peripheral Nerves, Lancet **2**:126 (Aug. 3) 1940.

sticks to the nerves and holds the stumps together. The plasma is freely permeable and during the subsequent days is dissolved away, although it remains long enough for a firm union to be established between the divided ends. The anatomic configuration of the fallopian aqueduct, lodged within the confines of the temporal bone, renders it most adaptable to this improved technic.

CHRONIC PROGRESSIVE DEAFNESS

Before one can regard as worth while an improvement in hearing following treatment for deafness, whether medical or surgical, there must be repeated audiometric examinations and voice tests, and they must show a sustained improve-

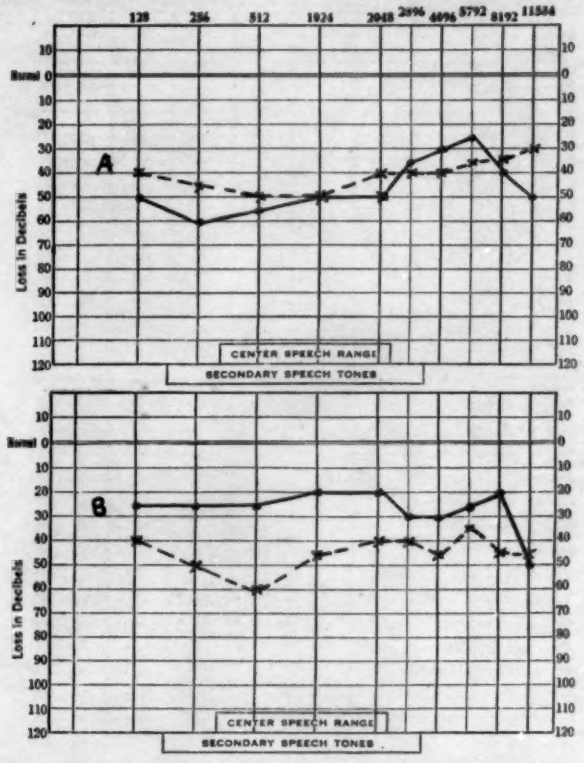

Chart 3.—Audiograms of Miss V. R., aged 28, taken (*A*) five days before and (*B*) twenty-four months after fenestration. She has regained her position as a stenographer.

ment of at least 10 decibels above the pretreatment level. To be of practical value to the patient, this improvement must include the conversational tones of 512, 1024 and 2048 double vibrations. Up to the time of the writing of this paper, it has generally been conceded that there is no medical treatment of proved value for otosclerosis. With this opinion Campbell [4] expressed agreement, in a review read before the American Otological Society in May 1942, a statement to which a few critics took exception. In support of surgical treatment is the possibility that this condition is Paget's disease involving the outer wall of the bony labyrinth. The only treatment which meets all the accepted requirements is a surgical measure

4. Campbell, E.: Fenestration of Labyrinth: A Report and Analysis of Operated Cases, Laryngoscope **52**:593 (Aug.) 1942.

called the fenestration, or window, operation. This procedure is based on the fact that the hearing defect in otosclerosis is due primarily to an obstruction to sound conduction, not in the middle ear but in the walls separating it from the peripheral terminals of the auditory nerve in the cochlea.

After Lempert,[5] of New York, presented his outstanding contribution, the endaural fenestration operation, before the American Otological Society, in May 1938, it was my privilege to visit Groningen, Holland, in July 1938, and to hear Sourdille, of France, read his paper on the surgical treatment of otosclerosis before the European Collegium. Holmgren, of Stockholm, Sweden, and Nager, of Zurich, Switzerland, discussed this paper. Later, in Sweden, I visited Stock-

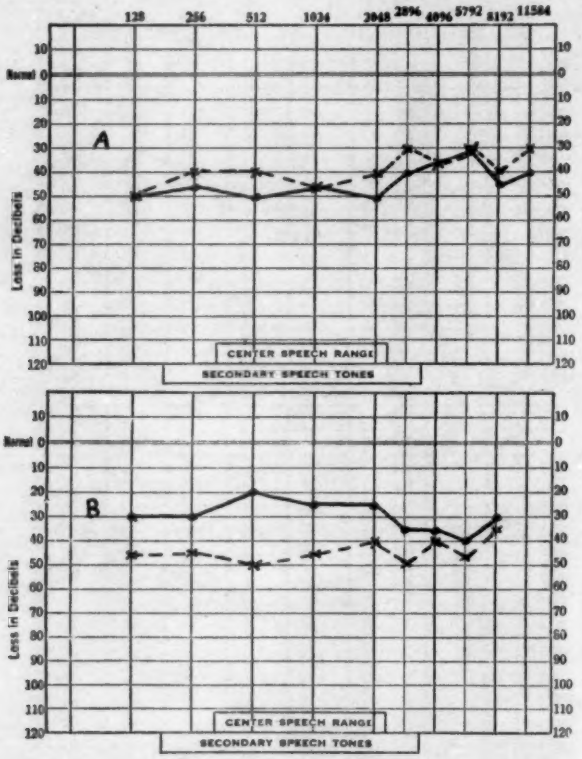

Chart 4.—Audiograms of Miss E. L., now Mrs. E. W., aged 28, taken (*A*) five days before and (*B*) three and a half years after fenestration. She is now married and has two children.

holm and Lund; at the latter university I learned much from the work on labyrinthology being done by Dohlman. On my return, I was afforded an opportunity in the department of otolaryngology of the University of Toronto to carry out experiments on the fenestration operation with monkeys and dogs. When Dohlman was in Toronto, in 1939, he saw some of my work and was flattering in his appreciative comments; he, too, endorsed the endaural technic.

I agree with the statement by Shambaugh Jr.[6] that Lempert has made a monumental contribution to otology in continuing the pioneer work of Passow, Bárány,

5. Lempert, J.: Improvement in Hearing in Cases of Otosclerosis: A New One Stage Surgical Technic, Arch. Otolaryng. **28**:42 (July) 1938.

6. Shambaugh, G. E., Jr.: Graduate Lecture Course, American Academy of Ophthalmology and Otolaryngology, 1941.

Jenkins, Holmgren and Sourdille and in devising a practical one stage operation. The procedure is one of the greatest progressive steps in the history of otology and offers the patient with otosclerosis the reasonable possibility of recovering much of his lost hearing.

With the perfection of technic, and from personal observations during the last four years, I have found that my best results were obtained in cases in which I removed the incus and made the fistula toward the ampulla of the horizontal semicircular canal, because I was able to mold and place the thinnest part of the tympanomeatal membrane over the fistula.

Recently, in November 1941, Lempert [7] again introduced a modification of his technic which he designated as the fenestra nov-ovalis operation, and in which,

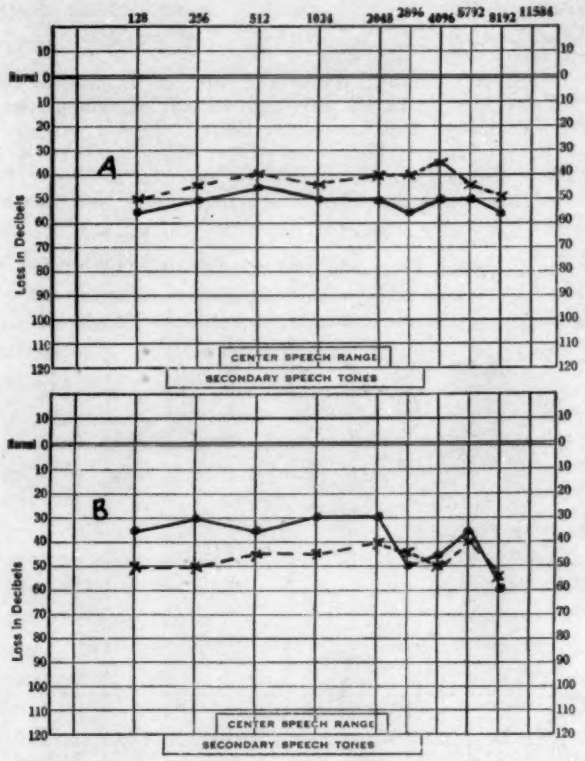

Chart 5.—Audiograms of Mr. A. C., aged 25, taken (*A*) two days before and (*B*) eighteen months after a revision, on May 3, 1940. The fistula has since remained open. Excessive scar tissue was removed at the second operation.

briefly, the fenestra is made practically in the dome of the vestibule. A discusser of a recent paper on this technic emphasized the modification as evidence of the yet unestablished infallibility of the fenestration operation. I take issue with this observation; an illustration in point is the improvement in technic of the nerve grafting operation, as described in the first part of this paper. Every good surgical technic undergoes continual stages of improvement and development. Why should the fenestration procedure be any exception to this fundamental principle of surgery? Experienced surgeons are continually modifying their procedures, but no one suggests they should cease operating. I have performed operations on over 75

7. Lempert, J.: Nov-Ovalis, Arch. Otolaryng. **34**:880 (Nov.) 1941.

patients and have carried out considerable experimental work on animals. My comments and observations are based on this experience, not on physiologic and academic inexactitudes.

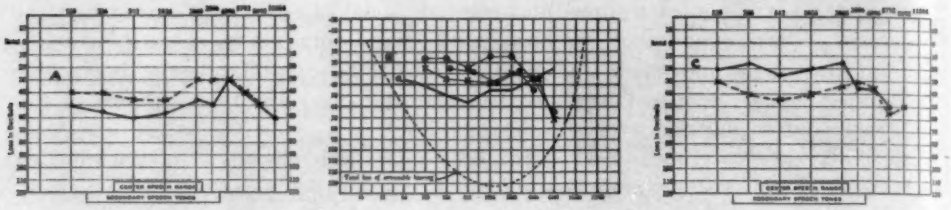

Chart 6.—Audiograms of Mrs. A. M. S., aged 35, taken (*A*) one day before, (*B*) two months after (by Dr. W. J. McNally of Montreal, Canada) and (*C*) six months after fenestration. The nov-ovalis technic was used.

In *B*, the line of hollow dots indicates the hearing loss, in decibels, in the right ear; the line of crosses, the hearing loss in the left ear, and the line of solid circles, hearing in the right ear with the left ear masked with an electric buzzer. Line *a* indicates the audiometric reading for a person with normal reading when tested with the D-80904 bone conduction receiver. For this patient, whisper was heard with the right ear at 5 feet (15.2 cm.), distinct conversation with the right ear at 10 feet (305 cm.) and whisper with the left ear at 2 feet (60 cm).

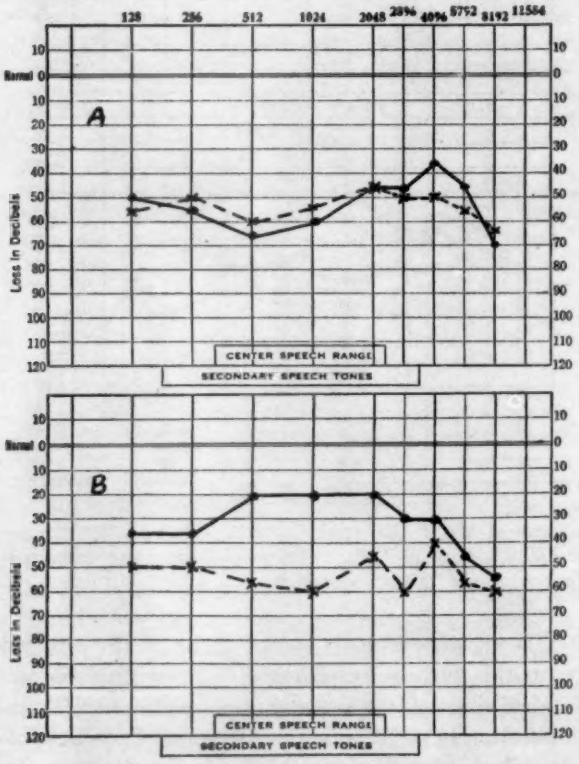

Chart 7.—Audiograms of Miss D. M. B., aged 40, taken (*A*) two days before and (*B*) twenty-six months after fenestration. The incus was removed. She hears better after fenestration than she did for years with a hearing aid.

Some preoperative and postoperative audiometric curves are shown in charts 1 to 7.

CONCLUSIONS

After the publication of Lempert's paper [8] in April 1941, I listed the following apparent reasons for failure in the endaural operation:

1. Failure to perform meticulously each successive step in the operation—one mistake is invariably fatal.

2. Selection of the improper type of case for operation. (I realize that one must not forget that deaf patients are eager to try any new method offering a semblance of hope. To hear, one must listen, which is a motor act that can be stopped by unswitching the dendrites, and as all deaf patients cease to listen after a while, their deafness is apparently increased thereby. While they are undergoing the long procedure which this treatment demands, they reeducate their listening ability, and no doubt in this way they regain some additional hearing.)

3. The precision required in making the fistula, coupled with the careful molding and placement of the flap over the fistula.

4. Failure to recognize damage to the membranous labyrinth at the time of fistulization.

5. Whether the operation which improves the hearing retards or inhibits the general progress of the disease remains for time alone to decide. The fact that a practical gain in hearing has remained in some cases for over four years tends to substantiate the belief that it may.

6. There is no question regarding the value of hearing aids. Great credit is due the acoustic engineers for the rapid strides they have made in the devising of new and improved aids. Here, I should like to utter a word of caution: The otologist should test and select the proper hearing aid for the patient. However, it has been my experience that a patient who has worn a hearing aid and who later submits to a successful fenestration operation will frankly state that he prefers the postoperative state of his hearing—"The voice he hears as a result of the surgical procedure seems normal."

I do not for a moment mean to say that all persons with otosclerosis should have this operation. The amount of deafness may not constitute a disability; rather it may be only an annoying discomfort. In many cases, if the loss is excessive, the patient may have relief from the operation.

To recapitulate, the one stage fenestration operation for otosclerosis offers for the first time a means of restoring hearing. The operation is indicated for patients with otosclerosis whose hearing loss is sufficient to constitute a handicap and who have an intact nerve of hearing for the conversational frequencies. The operation is technically intricate, delicate and time consuming, but if the procedure is correctly followed at each successive step a practical gain in hearing will be secured.

The operation is an entirely new procedure in otologic surgery, in which one mistake is invariably fatal, and not to be compensated for by nature, which treats the otologic surgeon so kindly in the case of "infectious diseases" of the temporal bone.

Dr. C. H. Best, professor of physiology at the University of Toronto, extended to me the use and privileges of his operating room for my work on monkeys.

Medical Arts Building.

8. Lempert, J.: Endaural Fenestration of the Horizontal Semicircular Canal for Otosclerosis, Laryngoscope **51**:330 (April) 1941.

Case Reports

VARICELLA VESICLE IN THE EXTERNAL AUDITORY CANAL

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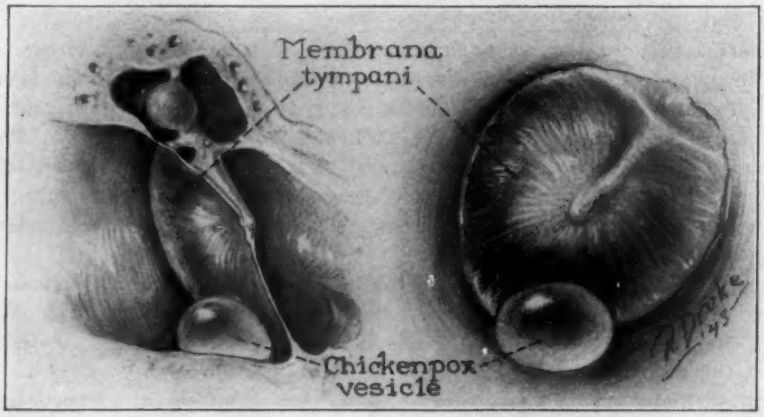
In a most comprehensive statistical analysis of the complications of varicella, Bullowa and Wishik [1] stated that some type of complication occurred in 133 (5.2 per cent) of a group of 2,534 cases of varicella encountered at the Willard Parker Hospital in the five years from Jan. 1, 1929, to Dec. 31, 1933 inclusive. Most frequent among the complications was otitis media, which occurred in 37 cases.

Although herpes zoster oticus is not unusual and although there is apparently a distinct relation between herpes zoster and chickenpox,[2] records of chickenpox involving the ear are scant.



Vesicle on floor of external auditory canal: lateral and anteroposterior views.

The purpose of this case report is to point out the possibility of involvement of the ear by the lesions of chickenpox and to stress the necessity for thinking of this possibility in the differential diagnosis of otitis media complicating varicella.

REPORT OF CASE

A girl aged 9 years had been found to have varicella; the prodromal symptom of malaise had been noted on May 21, 1941. The following day, May 22, a typical macular eruption was noted. The trunk was involved principally, but a few macules appeared on the face

1. Bullowa, J. G. M., and Wishik, S. M.: Complications of Varicella: Occurrence Among 2,534 Patients, Am. J. Dis. Child. **49**:923-926 (April) 1935.

2. Brain, R. T.: The Relationship Between the Viruses of Zoster and Varicella as Demonstrated by the Complement-Fixation Reaction, Brit. J. Exper. Path. **14**:67-73 (April) 1933. Bruusgaard, E.: The Mutual Relation Between Zoster and Varicella, Brit. J. Dermat. **44**:1-24 (Jan.) 1932. Stern, E. S.: Mechanism of Herpes Zoster and Its Relation to Chickenpox, ibid. **49**:263-271 (June) 1937.

and forehead. The temperature was 99 F.; the pulse rate was normal, and she did not appear especially ill.

On May 22 the child complained on awakening of a sense of fulness in the left ear, and later in the day mild otalgia developed. At this time there was a mixture of macules, papules and vesicles over the trunk, with a few scattered lesions on the face and extremities. Examination of the right ear revealed nothing abnormal. The left ear showed no sign of otitis. However, on the floor of the external auditory canal (figure), directly at its juncture with the tympanic membrane and obviously in contact with the tympanic membrane, a typical vesicle of chickenpox was noted. Examination of the nose and throat revealed nothing significant.

Since the child did not appear to be in great distress, no specific treatment was instituted, but the lesion was observed. If the child had been in great pain the vesicle would have been punctured carefully. By the following day the lesion had become pustular and smaller and had ceased to press on the tympanic membrane. The otalgia had entirely disappeared, as had the sensation of fulness in the ear. The lesion continued to dry up, and four days after it had been noted, had entirely disappeared, leaving a normal-looking external auditory canal and tympanic membrane.

UNUSUAL CYST OF THE MASTOID

D. F. Weaver, M.D., Detroit

Since a study of the literature for the past ten years indicates that cyst of the mastoid is rarely encountered, it is felt that the following case is of interest.

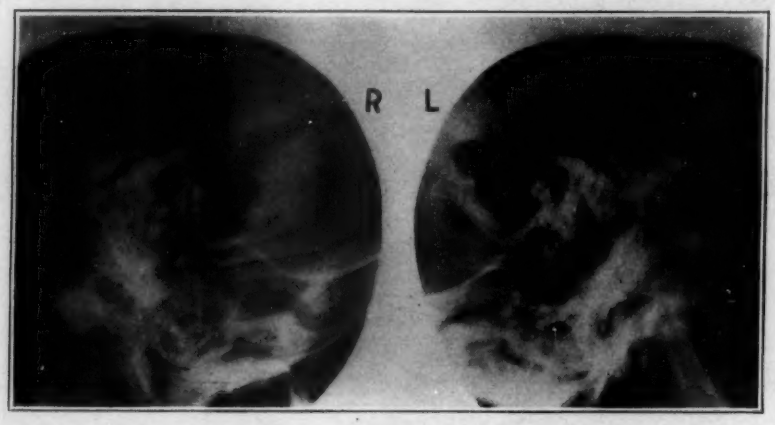
REPORT OF A CASE

A white man aged 27 was admitted to the Henry Ford Hospital on Feb. 24, 1942. He stated that he had had a discharge from the left ear for several months and troublesome dizziness for several days. There had been no pain. He had no previous history of aural disease.

Examination of the left ear revealed that the posterior wall of the canal bulged forward to such an extent that the membrana tympani could not be seen. A small amount of thin serous discharge was present.

Audiograms showed normal hearing in the right ear and a loss of between 20 and 30 decibels in the conversational range in the left ear. Bone conduction was practically normal.

Roentgenograms of the left mastoid gave the appearance of a postoperative condition. The right mastoid was normal (figure). The question of suppurative mastoiditis with complete breaking down of the cells arose, but in view of the atypical history and findings, a cyst of some type was thought of. Otherwise the history referable to the ears, nose and throat and the results of examination were without significance.



Roentgenograms of the mastoid regions. *R* indicates the right mastoid; *L*, the left mastoid.

General physical examination revealed no significant abnormalities. The neurologic signs were reported to be negative. The blood pressure was 122 systolic and 70 diastolic. The hemoglobin content was 14.9 Gm. per hundred cubic centimeters, and the leukocyte count was 6,400. The Wassermann reaction was negative, and urinalysis revealed nothing significant.

On February 27, with the patient under anesthesia induced with ethylene and cyclopropane, the cortex was removed from the left mastoid process and a large cystic cavity, involving the entire mastoid and extending well into the petrous pyramid, was observed. The cavity was apparently lined with a very thin, tissue-like membrane, and a large amount of sebaceous-like material was present, although the cavity was not filled with it. The material was readily removed with the suction tip. The lateral sinus had been completely uncovered throughout its extent in the mastoid. The remnant of the posterior bony wall of the canal was thin and extended only a few millimeters from the annulus tympanicus. It was also displaced forward, until the short approach to the membrana tympani was only 4 or 5 mm. in diameter. The facial ridge was extremely low. In the region of the mastoid

From the Division of Otolaryngology, Henry Ford Hospital..

antrum and the horizontal semicircular canal, the landmarks were so bizarre that it was impossible to determine definitely the location of these structures.

It was felt that under the circumstances one would be justified in not disturbing the middle ear and in not removing further bone in this region. A flap was made from the posterior membranous wall of the canal, similar to the flap that is made in the radical mastoidectomy. The cavity was packed through the ear with petrolatum gauze. The pack was removed on the fifth postoperative day, and the patient was dismissed from the hospital.

The pathologist [1] reported that microscopic examination of the sections of tissue presented showed keratohyaline strands. There was nothing to suggest the presence of cholesterol or an inflammatory reaction. Unfortunately, in the tissue presented to him an epithelium-lined section could not be found. Consequently, a certain amount of doubt exists as to the exact diagnosis. However, it was his opinion, since he thought the material examined was sebaceous, that the cyst was of either dermoid or sebaceous type.

The cavity drained through the ear for several months. At present, one year after the operation, the ear is completely dry. The cavity has become completely epithelized and can be inspected through a large opening over the proximal portion of the facial ridge. There has been no dizziness since the operation. The hearing is about 10 decibels better in the conversational range.

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1. Hartman, F. W.: Personal communication to the author.

Progress in Otolaryngology

Summaries of the Bibliographic Material in the Field of Otolaryngology

CHRONIC PROGRESSIVE DEAFNESS, INCLUDING OTOSCLEROSIS AND DISEASES OF THE INNER EAR

GEORGE E. SHAMBAUGH JR., M.D.
AND
FRANK WOJNIAK, M.D.
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The literature on chronic progressive deafness for 1941 includes important contributions on acoustic trauma and the use of prostigmine for deafness and an increased number of papers on the fenestration operation. As has been customary in this review, our editorial comments are in brackets, and only articles contributing something new are summarized. The literature is covered in the following order:

Otosclerosis
- Symptoms and pathologic features
- Treatment other than the fenestration operation
- The fenestration operation

Chronic adhesive deafness

Primary nerve deafness
- Traumatic nerve deafness
- Nerve deafness from drugs
- Nerve deafness from avitaminosis
- Nerve deafness from miscellaneous causes
- Treatment of nerve deafness
- Ménière's syndrome

OTOSCLEROSIS

Symptoms and Pathologic Features.—Holmgren [1] contributes an interesting article on the clinical symptoms in the external and the middle ear in cases of otosclerosis. The absence of cerumen in cases of far advanced otosclerosis was observed by Toynbee as early as 1860. Leesen found the auditory canal free from cerumen in 783 out of 912 cases of otosclerosis, while the British Otosclerosis Committee, in 1931, on the basis of answers to a questionnaire stated that cerumen was absent in 54 per cent of the cases. With the reduction in cerum, cutaneous irritability or sensitivity is reduced. Froeschels in particular emphasized this symptom and found the reduction to be proportionate to the severity of the deafness.

Abnormal wideness of the auditory canal, atrophy of the skin in the canal and abnormal transparency of the drum membrane have also been noted in cases of otosclerosis, and these observations are confirmed by the author.

Immobility of the manubrium, as determined with a Siegle speculum containing two parallel lines, is frequently present, with restricted mobility in still more cases and normal mobility relatively rare. This clinical observation was confirmed by Covell's histologic notation of distorted ossicular articulations in 95 per cent of cases of otosclerosis with fixation of the stapes.

Holmgren adds a new symptom to these previously observed symptoms, namely, absence of the vascular reflex. Thus, if the inner end of the canal and the

1. Holmgren, G.: Otosclerosis: Symptoms in the External and Middle Ear, Acta otolaryng. **29**:91 (March) 1941.

drum membrane are touched with a swab of cotton or strong suction and pressure are applied with the Siegle otoscope, a lively reflex hyperemia normally results. In cases of otosclerosis this reaction is reduced or entirely absent. Similarly, the cough reflex which is obtained when the deeper part of the canal is touched is often much reduced in the presence of otosclerosis.

Holmgren suggests that an increase in the intralabyrinthine perilymph pressure may cause pressure on the fenestra, resulting in immobility of the stapes, followed by gradual ankylosis and rigidity of other ossicles. The nutrition of the drum membrane, which normally is in constant vibration, is then affected, with resulting atrophy, decreased sensitivity and less cerumen in the adjacent canal. [During more than 300 fenestration operations, one of us (G. E. S. Jr.) has never observed any evidence of increased perilymph pressure, nor is there any histologic or clinical evidence to support Holmgren's hypothesis.]

Peyser,[2] in a study of 736 otosclerotic ears, found that the triad of lack of cerumen, absence of the cough reflex and absence of the vascular reflex of Holmgren was present in 30 per cent. In only 2.7 per cent of these ears was there complete absence of all three symptoms. The triad increased in frequency in proportion to the decrease in mobility, and the incidence was much higher when the disease had lasted longer than five years.

Hagens [3] reports the occurrence of otosclerosis in a pair of identical twins. The father had normal hearing, but the mother had otosclerosis and came from a family of 10 children, of whom only 2 had normal hearing. The onset occurred at the age of 17 years in one twin and a few years later in the other. The twin with the earlier onset became pregnant a year after the appearance of the disease, with a noticeable increase in her hearing defect following this pregnancy. The twin with the later onset of deafness had never been pregnant, and her defect was the lesser of the two. Both twins had been subject to frequent colds, an etiologic factor noted by both Fowler and Shambaugh Jr., and both had disliked fruits and vegetables and had eaten an excess of starches and sugar. [Otosclerosis has now been reported in 6 pairs of identical twins, and in every instance both twins were affected, evidence of the importance of the hereditary factor in this disease.]

Jones [4] reviews the various gross pathologic changes that have been observed in the otic capsule as a result of otosclerosis. These lesions include foci in the semicircular canals, thickening of the lateral labyrinthine wall, with narrowing and obliteration of one or both windows, tumor-like protuberances into the labyrinth and internal auditory meatus and obliteration of the facial canal. Labyrinthine atrophy is a part of the typical picture of otosclerosis. The author concludes that the concept of fistulization of the semicircular canals for improvement of hearing in patients with otosclerosis is misleading, since there may be degeneration of the acoustic nerve and the patient may not be benefited. [It is, of course, necessary to select patients for the fenestration operation; not every patient with ankylosis of the stapes is suitable, the condition of the nerve of hearing being a determining factor.]

Several interesting articles appeared in 1941 on the association of otosclerosis with blue sclerotics and brittle bones (osteogenesis imperfecta). Riddell [5] traces the largest pedigree for this condition thus far worked out, consisting of 208

2. Peyser, A.: Weiteres über Symptome der Otosklerose am äusseren und Mittelohr, Acta oto-laryng. **29**:167 (May) 1941.

3. Hagens, E. W.: Otosclerosis in Identical Twins, Arch. Otolaryng. **34**:583 (Sept.) 1941.

4. Jones, M. P.: Otosclerosis with Unusual Pathological Findings, Laryngoscope **51**:714 (July) 1941.

5. Riddell, W. S. B.: Pedigree of Blue Sclerotics, Brittle Bones, Deafness and Color Blindness, Ann. Eugenics **10**:1 (April) 1940.

persons. No conclusion or discussion is given, but the strong hereditary tendency of this syndrome is evident.

Corboy [6] states that 60 per cent of patients with blue sclerotics have fragilitas ossium, 60 per cent have otosclerosis and 44 per cent have all three defects. The blue color of the sclera is due to lack of opaqueness, which allows the uveal pigment to shine through. The author presents the case of a mother, 2 sons and a daughter with this syndrome (van der Hoeve's syndrome); all 4 had low calcium and phosphorus levels of the blood (7.53 to 8.01 and 1.96 and 2.19 mg. per hundred cubic centimeters respectively).

Riesenman and Yates [7] state that osteogenesis imperfecta (fragilitas ossium) is a hereditary mesenchymal hypoplasia and is transmitted as a dominant mendelian factor. The most constant clinical feature is the blue sclerotics, found in 100 per cent, with brittle bones present in 65 per cent and otosclerosis in 43 per cent. Of 255 members of 7 family groups, 91 had the disease.

[The frequent occurrence of otosclerosis in this syndrome was first noted in 1912 (Adair-Dighton), and pathologic examination of the ears in a case of the disease, first made by Nager in 1926, showed typical advanced otosclerosis. A clue to the causation of otosclerosis may be found through studies of this interesting and unique syndrome.]

Treatment Other Than the Fenestration Operation.—Garnett Passe,[8] having observed decided reduction in tinnitus and improvement in hearing in a patient with otosclerosis after withdrawing 80 cc. of spinal fluid and replacing it with 60 cc. of air for ventriculographic examination, carried out the same procedure on 6 more patients with otosclerosis, 4 of whom had tinnitus. All the patients experienced a considerable degree of improvement in hearing with temporary subsidence of tinnitus. For twenty-four to forty-eight hours after this treatment the patient has a severe headache, and he is fit to return to work on the third or fourth day. No audiometric record was available for the reported cases, the improvement in hearing being measured by tests with the spoken voice. The author does not specify the amount of the improvement, nor does he offer any rationale for this treatment. [As previously stated in this review, we cannot accept as of proved value any treatment for deafness not substantiated by adequate audiometric tests. It is to be hoped that the author will be able to prove his claims audiometrically.]

Guggenheim, Gunther, Goodhill and Irvine [9] state that surrounding the focus of otosclerotic bone there is usually an area of decalcification, or halisteresis; this becomes filled with osteogenic bone marrow in which new, abnormal bone forms, so that the size of the focus is increased. With dietary regulation and the administration of dicalcium phosphate (Ca H Po_4), vitamins, insulin, liver extract and other preparations, a series of patients with chronic otosclerosis have had no further loss of hearing over a period of four years. Some of them have shown an improvement in hearing, presumably the result of the administration of thiamine hydrochloride, nicotinic acid, insulin and liver extract. To prove the possibility that halisteresis in the aural capsule may be reversed, ammonium chloride was fed to white rats, with

6. Corboy, P. M.: Van der Hoeve's Syndrome: Four Occurrences in Three Generations of One Family, Am. J. Ophth. **24**:57 (Jan.) 1941.

7. Riesenman, F. R., and Yates, W. M.: Osteogenesis Imperfecta: Its Incidence and Manifestation in Seven Families, Arch. Int. Med. **67**:950 (May) 1941.

8. Garnett Passe, E. R.: Cerebrospinal Puncture and Air Replacement for Otosclerosis, J. Laryng. & Otol. **56**:389 (Nov.) 1941.

9. Guggenheim, L. K.; Gunther, L.; Goodhill, V., and Irvine, M.: Reversal of Halisteresis in Mammalian Ear, Arch. Otolaryng. **43**:501 (Sept.) 1941.

production of halisteresis in the otic capsule. When ammonium chloride was removed from the diet and one high in calcium was given, the halisteresis was checked and osseous reconstruction occurred. [The authors' belief that growth of the otosclerotic focus is preceded by local halisteresis is not shared by all histologists. If by dietary management the progress of otosclerosis can actually be arrested and in some cases the hearing improved, let complete audiometric data be published substantiating this claim, such as are now available for treatment by fenestration.]

The Fenestration Operation.—In the literature for 1941 the number of articles dealing with the fenestration operation is larger than ever before.

Lempert published two papers. The first [10] presents his results in 150 cases in which a fenestra was made in the horizontal semicircular canal. In 39 cases revision was undertaken because improvement was not obtained or maintained. The surgical accidents were: (1) fracture of the bony fallopian canal, with temporary facial paresis, and (2) puncture of the lateral sinus, in 2 cases. In these 2 cases, however, bleeding was controlled and the operation completed; convalescence was uneventful.

The results of operation in this series were as follows: permanent restoration of practical physiologic hearing, 93 cases; excellent improvement in conversational hearing, but not sufficient to reach the level of practical hearing, 11 cases; further impairment of hearing, 14 cases, and no improvement in hearing, 32 cases.

Closure of the fenestra, when it occurred, was usually complete within three to ten weeks after operation. If signs of beginning loss of hearing in the fistula test were not apparent at the end of eight weeks, the fenestra remained permanently open.

The author states that his success in maintaining a patent fistula, with permanent improvement in hearing, depends on the following procedures: (1) mobilization of the perilymph for air-borne sound by creation of a fenestra; (2) care in covering the fenestra with a membrane to prevent infection; (3) reconstruction of the tympanic air space so that a minute air space is left between Shrapnell's membrane and the fenestra, the incus preventing adhesion of Shrapnell's membrane to the margins of the fenestra; (4) complete removal of the endosteum, without injury to the membranous labyrinth, and (5) prevention of excessive postoperative tissue reactions by the use of the endaural approach. Lempert concludes that there is today sufficient evidence that the progression of the hearing loss in cases of otosclerosis is retarded, or perhaps even checked by fenestration.

In his second article Lempert [11] summarizes his results in 300 cases of fenestration of the external semicircular canal over a period of three and a half years. Closure of the fenestra, partial or complete, occurred in 88 cases, while in 212 cases the fistula remained open. A second operation in 72 cases revealed closure by bone or fibrous tissue, which in some cases showed bony metaplasia.

To diminish the chances of closure, the author began to make the fistula in the dome of the vestibule anterior to the ampullated end of the horizontal canal, where the perilymph space is wider than in the horizontal canal, so that a larger window is permitted. Because this new fenestra is close to the fenestra ovalis and communicates directly with the scala vestibuli, the name "fenestra nov-ovalis" is given it.

The technic of the fenestra nov-ovalis operation is described in detail, with illustrations. It consists essentially in the same steps as does Lempert's original

10. Lempert, J.: Endaural Fenestration of the Horizontal Semicircular Canal for Otosclerosis: Indications, Technique, Observations as to Early and Late Post-Operative Results, Laryngoscope **51**:330 (April) 1941.

11. Lempert, J.: Fenestra Nov-Ovalis, Arch. Otolaryng. **34**:880 (Nov.) 1941.

operation, but with removal of the incus, after which the fistula is made in the dome of the vestibule. The edges of bone are shaved with a dental spoon to remove the cambium layer; any bone dust is washed out by means of a rubber bulb syringe and physiologic solution of sodium chloride, and the endosteum is removed with a special needle, with exposure of the utricle. Shrapnell's membrane can now be made to cover the fenestra in every case.

In every one of 75 cases in which this new technic was used, covering a period of eighteen months, the window has remained open, and in no case are there signs of impending closure. In 3 cases the window was inspected two to five months after operation, and in none was there the slightest sign of beginning regeneration of bone or formation of connective tissue.

Audiometric data for 8 patients operated on by the new technic are summarized in the following tabulation:

Case No.	Time After Operation, Mo.	Change in Hearing for Speech in Ear Operated On, Db.	Change in Hearing for Speech in Intact Ear, Db.
1	17	+ 13	— 5
2	14	+ 42	— 3
3	12	+ 35	— 2
4	10	+ 14	— 8
5	10	+ 20	0
6	8	+ 32	+ 12
7	8	+ 23	— 2
8	6½	+ 28	+ 13

Audiograms showing late improvements in hearing following operation by his original technic are also given; the data are summarized as follows:

Case No.	Time After Operation	Change in Hearing for Speech in Ear Operated On, Db.	Change in Hearing for Speech in Intact Ear, Db.
1	3 yr. 10 mo.	+ 20	— 5
2	3 yr. 3 mo.	+ 23	+ 3
3	2 yr. 8 mo.	+ 26	+ 3
4	3 yr. 3 mo.	+ 22	— 2
5	2 yr. 11 mo.	+ 22	+ 7
6	3 yr. 1 mo.	+ 45	— 7
7	2 yr. 5 mo.	+ 30	— 2
8	2 yr. 4 mo.	+ 26	+ 16
9	1 yr. 7 mo.	+ 42	+ 2
10	1 yr. 10 mo.	+ 25	+ 7

While we cannot subscribe to all of Lempert's statements—for example, his assertion that the utricle is brought into view by the nov-ovalis technic, that Shrapnell's membrane always reaches the fistula or that a minute air space remains between the covering membrane and the fenestra—nevertheless his results are impressive, and his fenestra nov-ovalis technic may represent an important advance in the development of the surgical treatment of otosclerosis. However, since Lempert does not give his complete results, the poor with the good, it is not possible from his report to conclude that the fenestra nov-ovalis technic offers an advantage. The number of decibels gained in the 8 cases in which the fenestra nov-ovalis technic was used and for which audiograms are presented is about the same as obtained in the 10 reported cases in which the original technic was employed.

Particularly interesting and significant are the cases in which improvement in hearing is still present two and three years, and in 1 case three years and ten months, after operation. It is increasingly probable that in at least some of these cases the fistula will remain open permanently, with permanent improvement in hearing.

During 1941 Campbell [12] published several reports of his results with the fenestration operation. Analysis of 15 cases in which hearing improvement had been partly or completely lost showed that this loss always occurred within the first five months after operation and that thereafter the hearing remained stationary. The author suggests that eight months be taken as the postoperative period after which the end result may be assessed. In 25 of 53 cases in which operation had been performed, covering a period of three years, the postoperative period was longer than eight months and recent audiometric tests had been made. Complete charts containing all the important audiometric and clinical data are presented.

Case No.	Time After Operation	Change in Hearing for Speech in Ear Operated On, Db.	Change in Hearing for Speech in Intact Ear, Db.
1	2 yr. 11 mo.	+34	+ 3
2	1 yr.	—18.3	— 6.7
3	2 yr. 8 mo.	—24.4	—11.3
4	4 mo.	— 5.3	— 4.2
4 (revision)	2 yr. 2 mo.	+ 6.8	+ 4.4
5	2 yr. 4 mo	— 6	+15
6	2 yr.	+16.6	—10
7	1 yr. 11 mo.	—26	— 0.5
8	1 yr. 2 mo.	+25	— 2.3
8 (revision)	13 mo.	—10.7	— 2.4
9	1 yr. 11 mo.	—14.7	—20.3
9 (other ear)	3 mo.	+22.3	+ 8.4
10	1 yr. 4 mo.	0	— 3.4
10 (revision)	7 mo.	+ 8.3	0
11	2 yr.	+20	+ 1
12	1 yr. 9 mo.	+21.3	— 1
13	1 yr. 11 mo.	+ 4	— 5.6
14	1 yr. 8 mo.	—16.6	+ 3
15	1 yr. 9 mo.	+ 2.6	+ 1.7
16	1 yr. 3 mo.	— 0.7	— 4.3
17	1 yr. 8 mo.	+15	+ 1.3
18	1 yr. 3 mo.	+14.7	+ 0.3
19	1 yr. 1 mo.	—35.7	— 7
20	1 yr. 6 mo.	— 2.3	+ 5
21	5 mo.	+20	+ 3
22	1 yr. 3 mo.	+19	+ 0.3
23	5 mo.	—30	— 1.6
24	5 mo.	— 5	— 6.7
25	10 mo.	+22	— 1.3
26	9 mo.	+ 0.4	+ 1.7
27	10 mo.	+35.6	+ 5.7
28	8 mo.	— 0.6	+ 0.3
29	3 mo. 3 wk.	+26	+ 6.7
30	4 mo.	+14	— 8.7

	No. of Cases
Audiometric tests made 8 mo. or more after operation	25
Hearing improved (more than 10 db. gain for speech)	10
Hearing loss greater (more than 10 db. loss)	6
Hearing unchanged (within 10 db.)	9

Of the cases in which hearing was unchanged or the loss greater, 9 were classified as unsuitable for operation, old radical cavities having been present in 3 and poor bone conduction in 6.

12. Campbell, E. H.: Further Experiences in Fenestration of the Labyrinth in Chronic Progressive Deafness: Report of Cases and an Analysis of Results, Ann. Otol., Rhin. & Laryng. **50**:182 (March) 1941; Fenestration of the Labyrinth in Chronic Progressive Deafness: An Analysis of Results Obtained, South. M. J. **34**:13 (Jan.) 1941; The Evaluation of the Fenestration Operation for Otosclerosis, Laryngoscope **51**:1009 (Nov.) 1941.

Campbell differs from Lempert in that he does not believe that Shrapnell's membrane can reach the fistula in most cases; he believes that the endosteum should not be removed and that the postaural approach is better than the endaural. He has had no serious complications and no fatalities. He points out that the highest improvement in hearing as a rule is not retained, but becomes somewhat reduced, owing to partial closure of the fenestra.

He concludes that the labyrinth fenestration operation performed in accordance with the principles of Sourdille offers a means by which the hearing in certain cases of chronic deafness may be improved. This improvement is retained to a degree that is practical in a sufficiently high percentage of cases to be a useful therapeutic procedure. [Campbell, again, is to be complimented on his complete report of bad results with the good, as well as on his good percentage of lasting and significant hearing gains. The number of decibels of improvement in Campbell's cases in which the results were successful compares favorably with the gains reported by Lempert, considering that Lempert presumably selected his 18 best cases.]

Shambaugh Jr.[13] describes an irrigating apparatus to be used while making the fenestra, in order that every particle of bone dust may be washed away. The apparatus is simple, consisting of two silver tubes that fit onto a self-retaining endaural retractor, the physiologic solution of sodium chloride flowing in at one end of the cavity through one tube and being removed at the other end by suction through the second tube.

Of 31 patients operated on over a period of two years and two months, 6 of whom had revisions and 3 operations on both ears, 26, or 84 per cent, have a hearing gain of more than 10 decibels for speech, this gain varying from 11 to 37 decibels. Eight of these patients had previously worn a hearing aid, and each one states that he now hears better than he could with the aid. The author's experience has been that a fistula which is open, with sustained improvement in hearing, three months after operation will remain permanently open, but he qualifies this statement by saying that with further experience it may need to be modified.

Ersner and Myers[14] report 38 cases in which the Lempert fenestration technic was employed, with a description of results in 17 of them.

Case No.	Time After Operation	Change in Hearing for Speech in Ear Operated On, Db.	Change in Hearing for Speech in Intact Ear, Db.
1	28 mo.	+ 20 (approximate)	0 (approximate)
2	9 mo.	+ 12 (approximate)	+ 4 (approximate)
3	9 mo.	+ 20	— 10
4	6 mo.	+ 24	— 7
5	Temporary improvement only		
6	No improvement		
7	Temporary improvement only		
8	Temporary improvement only		
9	Temporary improvement only		
10	Temporary improvement only		
11	Temporary improvement only		
12	Temporary improvement only		
13	Temporary improvement only		
14	Temporary improvement only		
15	Temporary improvement only		
16	Temporary improvement only		
17	Temporary improvement only		

13. Shambaugh, G. E., Jr.: Surgical Treatment of Otosclerosis, Wisconsin M. J. **40**:488 (June) 1941.

14. Ersner, M. S., and Myers, D.: Surgical Treatment of Deafness: Report of Experiences with the Endaural Labyrinthine Fenestration, Ann. Otol., Rhin. & Laryng. **50**:206 (March) 1941.

In 2 cases postoperative labyrinthitis resulted in a dead labyrinth on the side of operation. In 1 case pronounced vertigo and nystagmus on the patient's lying down developed one year after operation. In several patients there was further loss of hearing to far below the preoperative level.

The authors conclude that the tendency for the fistula to close is a factor over which at present there is no control. They believe that an increase in hearing that does not bring the hearing level to within the 35 decibel limit for speech is unimportant. They are encouraged by their success in 4 cases, for there is at present no other therapy which could accomplish as much. [These authors are to be complimented on their complete report of results, the bad with the good. Creating a false impression of results by glossing over the failures can only impede the development of this new therapy for the deafness of otosclerosis.]

Ersner and Myers [15] state that osteogenesis and closure of the fenestra continue to present the perplexing problem in fenestration of the labyrinth. To determine whether heat produced by the drill is a factor in inhibition of bone regeneration, the authors employed an electrocouple to measure the change in temperature when a fenestra was made in the horizontal semicircular canal in a cadaver. The maximum degree of change was 12.6 degrees (F). Application of the finishing burr once to twenty times to the mastoid cortex of the anesthetized cat resulted in a maximum change of 3.5 degrees (F.). When the same burr was applied to the promontory of this cat, the intralabyrinthine rise in temperature was 3.1 degrees (F.).

In contrast to these slight increases, cauterization or electrocoagulation of bone increased its temperature 40 to 50 degrees (F.) The authors conclude that heat produced in making a fenestra in the horizontal semicircular canal is not a factor in inhibition of osteogenesis, since the rise in temperature is too slight. While electrocoagulation or cauterization results probably in enough heat to retard or inhibit osteogenesis, the increased temperature would, no doubt, have a detrimental effect also on the membranous labyrinth. [That the highest proportion of patent fistulas with lasting hearing improvement thus far reported in the literature followed the use of constant irrigation, which would prevent any local rise in temperature, is further evidence that local heat is not a factor in the permanency of the fistula made by Lempert's technic.]

Kopetsky [16] believes that the diagnosis of otosclerosis is almost impossible to make clinically in the present state of knowledge. He maintains that the adding up of successes for a given surgical technic hardly rates serious consideration as a scientific contribution to the advancement of knowledge on this subject. He warns that one should avoid comparing the results of one surgeon with those of another, as each operator is probably using a different procedure and the patients are differently selected.

Kopetsky lists 27 operations (26 patients), in which he used six different technics, with audiometric records for 4 of the patients.

Case No. and Technic	Time After Operation	Change in Hearing for Speech in Ear Operated On, Db.	Change in Hearing for Speech in Intact Ear, Db.
1 (fenestration in a radical cavity)	Improvement, lasting 5 mo.	+25	?
2 (Sourdille technic)	Results not stated	+15	?
3 (postaural approach, incus removed)	Results not stated	+37	+13
4 (skin flap sloughed)	21 mo.	+23	+12

15. Ersner, M. S., and Myers, D.: Does Heat Created by the Dull Dental Burr During Labyrinthine Fenestration Inhibit Osteogenesis? Arch. Otolaryng. **34**:121 (July) 1941.

16. Kopetsky, S. J.: History and Present Status of Operations on the Labyrinthine Capsule for Otosclerosis, Surg., Gynec. & Obst. **72**:466 (Feb.) 1941.

The author concludes that as yet no sure way has been devised of making a fenestra that can reasonably be expected to remain open permanently. The greatest possible care must be exercised in making promises of what will be accomplished by these operations. [With the last statement we are in complete accord. The patient deserves to be told exactly the chances of success or failure and the risks involved in the particular technic at the hands of the particular surgeon, for the fenestration operation is distinctly an elective procedure, with results that appear to vary greatly with the technic employed.]

Kend [17] administers a diet free from vitamin D, high in calcium and low in phosphorus, with vitamin D–depressing foods and bovine ovarian extract for two weeks before and four weeks after operation, in order to depress the periosteum and prevent new bone formation. An unlooked-for result has been an almost dry postoperative cavity during the entire period of epidermization.

The surgical technic employed by Kend consists in the postaural approach, with enlargement of the meatus by a curved incision, the flap thus formed being sutured backward and held in place with balls of poroform mesh gauze. The author states that this technic reduces the operating time, shortens the convalescence and leaves a good cosmetic result. The results will be presented in a subsequent report. [It is difficult to see how the making of two skin incisions instead of one should yield the advantages cited by the author, when the endaural incision can give adequate room and exposure. The author's failure to state his results detracts from the usefulness of this report.]

Katz [18] points out inconsistencies in Lempert's first article on the fenestration operation, including a case in which hearing in the intact ear gained more (20 decibels) than hearing in the ear which was operated on (17 decibels). The postoperative period of observation in about half the cases was four weeks or less, the average period being one and a half months, which is entirely insufficient for a final judgment of results. The author criticizes as dangerous the exposure of the dura of the middle fossa, since it makes the procedure essentially an operation on the brain. He believes that the improvement in hearing observed immediately after fenestration is due to decreased labyrinthine pressure, permitting an increased blood supply to the cochlea.

In conclusion, the author suggests that a committee of experts examine the patient before and after operation in order that a subjective judgment of results be avoided. [With this last suggestion we are in hearty accord, if there are still those who have not yet had the opportunity of making such examinations. We, and I am sure other surgeons doing this work, would be glad to have any or all of our patients examined before and after operation by any qualified otologist or committee of experts, if there are those who, despite the now numerous published reports, still doubt the validity of the recorded results.]

Lathrop [19] reports 7 cases in which the Lempert technic was used. In 2 cases the operation was too recent for appraisal of results; in 4 cases improvement followed, and in 1 case the hearing loss was greater (decibel change not specified). Audiograms in 1 case showed an average gain in hearing for speech of 27 decibels two months after operation.

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17. Kend, L.: The One-Stage Fenestration Operation Using the Postaural Approach, Laryngoscope **51**:37 (Jan.) 1941.

18. Katz, B.: Limitations of Surgery in Otosclerosis: A New Theory for the Immediate Improvement of Hearing in Fistula Operations, Laryngoscope **51**:246 (March) 1941.

19. Lathrop, F. D.: Surgical Treatment of Otosclerosis, Lahey Clin. Bull. **2**:103 (April) 1941.

Spake [20] asserts that no treatment for chronic progressive deafness except the surgical has given substantial improvement in hearing as shown by audiograms, and that the future treatment of this type of deafness is surgical. He emphasizes that no otologist, no matter what experience he has had, should attempt the fenestration operation without special training in the procedure; otherwise, the operation will fail, and the future progress of the work will be handicapped. [The author does well to emphasize that no otologist should attempt the fenestration operation without previous and adequate training in the technic, for in no other way can satisfactory results be hoped for.]

Canfield [21] discusses the indications for fistulization of the otic capsule, emphasizing the importance of the psychologic aspects in each case. Three cases are presented. In a suitable case a gain of 17 decibels for the speech frequencies was present one month after operation, and in 2 cases which were not suited for operation no gain resulted.

CHRONIC ADHESIVE DEAFNESS

Crowe and his associates [22] continue to emphasize the importance of lymphoid hyperplasia in the nasopharynx as a cause of impaired hearing in children. They find that adenoids recur in more than 75 per cent of children whose adenoids are removed before puberty and that this recurrence often interferes with the ventilating function of the eustachian tubes and leads to an insidious, painless type of progressive deafness, with more impairment for high tones than for low ones. The partial obstruction of the tubes leads to hyperemia of the mucosa of the middle ear, followed by hypersecretion of mucus and, finally, a myxomatous state or formation of fibrous tissue, which interferes increasingly with the mobility of the ossicles. The first otoscopic evidence is retraction of Shrapnell's membrane, and the first hearing loss noted is from 8,000 to 16,000 vibrations.

Treatment consists in surgical removal of the tonsils and adenoids, followed by irradiation for reduction of lymphoid tissue that cannot be reached surgically and occasional inflation of the tubes. Sometimes it is advisable to do a paracentesis and with a catheter blow out the secretions into the external canal. General hygienic measures should be instituted.

The authors believe that approximately 50 per cent of cases of deafness in adults can be prevented if school children are examined for the early loss for high tones and are appropriately treated. [In a study of members of the League for the Hard of Hearing made by one of us (G. E. S. Jr.) in 1933, only 10 per cent were found to have an adhesive type of deafness, and many of these had had chronic suppurative otitis media. It is our belief, contrary to that of Crowe and his group, that while tubal occlusion is the most important cause of impaired hearing in children, and an important cause of temporary impairment in adults, it rarely, if ever, leads to progressive and permanent changes sufficient to cause severe and handicapping permanent deafness in adults. The process is essentially a reversible one, and removal of the hypertrophied lymphoid tissue has resulted in restoration of essentially normal hearing in a case of deafness of this sort of twenty years' duration.]

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20. Spake, L. B.: The Surgical Treatment of Deafness, J. Kansas M. Soc. **42**:220 (May) 1941.

21. Canfield, N.: The Type of Deafness Which Responds to Fistulization of the Otic Capsule, Tr. Am. Laryng., Rhin. & Otol. Soc. **46**:450, 1940.

22. Crowe, S. G., and Burnham, C. F.: Recognition, Treatment and Prevention of Hearing Impairment in Children, Ann. Otol., Rhin. & Laryng. **50**:15 (March) 1941.

Polvogt [23] has found that some children with loss for high tones regained their hearing after the removal of large tonsils and adenoids and that those who did not improve frequently had lymphoid tissue around the orifices of the eustachian tubes. He concludes that long-continued partial obstruction of the eustachian tubes during childhood accounts for the most common type of middle ear deafness in adults.

Schenck [24] examined histologically lymphoid tissue removed from the nasopharynx in the neighborhood of the eustachian orifices before and after irradiation in 22 children between the ages of 6 and 12 years who had previously had their tonsils and adenoids removed, but who at the time of the study presented hyperplasia of Waldeyer's ring with hearing loss. The irradiation was carried out in three ways: 200 r of roentgen radiation to each eustachian orifice for three successive days; application of radium, alternating with roentgen irradiation, at weekly intervals for four treatments, and application of radium once a week for four treatments. Biopsy specimens taken two and four weeks after completion of the treatment showed little alteration in the adult lymphocytes but notable degeneration of the young lymphocytes in the germinal centers. The lymphatics, which had been largely blocked by proliferation of fibroblasts and endothelium before irradiation, showed remarkable disappearance of these elements, with opening and widening of the lymph channels. Minute hemorrhages were observed about and within the lymphoid nodules. Biopsy at six months revealed diminution in the amount of fibrous tissue and absence of the evidence of chronic inflammation seen before irradiation. The lymphoid follicles showed beginning regeneration, an indication that treatment should be repeated.

Hearing was improved on the average as much as 20 decibels for the 128 to 16,384 frequencies in 8 of the 15 patients who completed their course of treatment. No significant difference was observed in the histologic changes for the three technics. Schenck suggests that increased function of the lymphatics in and about hyperplastic lymphoid tissue exhibiting chronic inflammatory changes may be the chief result of this therapy. [This paper is an excellent and valuable contribution to the mechanism of the improvement in hearing following irradiation of the nasopharynx.]

Fowler [25] advises that an audiometric test be made on every child on entrance to school and that the examination be repeated every six to twelve months if impairment of hearing is suspected.

Mao and Hughson [26] examined 331 children with loss of hearing which required their being educated in the Pennsylvania School for the Deaf, to determine whether or not the eustachian tubes were normally patent. Autoinflation was employed, with a water manometer to measure the pressure required, 60 cm. of water being regarded as adequate to open the normal tube. Normally patent tubes were found in 206 children; in the remainder the tubes failed to inflate normally. However, the incidence of lymphoid tissue around the orifices was no higher in tubes with impaired inflatability than in normal tubes, and since all the children had less than useful hearing, the authors believe that there is no relation between the patency of the eustachian tubes and profound deafness in children.

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23. Polvogt, L. M.: The Prevention and Treatment of Deafness in Children by Irradiation, Tr. Am. Laryng., Rhin. & Otol. Soc. **46**:348, 1940.

24. Schenck, H. P.: The Influence of Nasopharyngeal Hyperplasia on the Ear: Histologic Examination of Hyperplastic Lymph Follicles After Irradiation, Laryngoscope **51**:780 (Aug.) 1941.

25. Fowler, E. P.: Deafness in Children: Early Detection, Management and Treatment, New York State J. Med. **41**:1543 (Aug. 1) 1941.

26. Mao, C. Y., and Hughson, W.: Patency of the Eustachian Tube in Relation to Profound Hearing Loss in Childhood, Arch. Otolaryng. **33**:824 (May) 1941.

Rhegvad [27] disagrees with Holmgren that secretory catarrh is the result of negative pressure. He thinks, rather, that there are several stages between suppurative otitis media and secretory catarrh, all being the result of local inflammation. To substantiate this he asserts that when paracentesis of the ear is done in cases of catarrhal deafness he has never seen bubbles rise in the fluid in the middle ear. [Contrary to the author's experience, we have many times observed bubbles enter the middle ear when it was perforated for relief of secretory otitis. Our observations lead us to conclude that while secretory otitis occasionally follows an inflammatory process in the middle ear, in most cases it is the result of negative pressure, without local inflammation in the ear.]

Van Dishoeck [28] used a "pneumophone" to determine the presence or absence of altered intratympanic pressure. This device consists of a rubber tube that fits into the external auditory meatus, into which a constant sound is introduced and in which the pressure can be altered by means of a rubber bulb and measured by a manometer. The apparatus works on the principle that the hearing is best when the intratympanic pressure equals the pressure in the meatus. The author found that complete obstruction of the eustachian tube causes a negative pressure of about 50 cm. of water. In cases of tubal catarrh due to head cold the negative pressure does not exceed 30 cm. Only about 20 decibels of the hearing loss in cases of tubal catarrh is caused by the negative pressure, the rest being due to some other factor, such as the pressure of fluid against the windows.

Johnson [29] studied 46 patients who had undergone submucous resection to determine the relation between nasal obstruction and impairment of hearing. The patients ranged in age from 14 to 54 years. Audiograms were taken before and about nine months after operation on the septum. The hearing was normal in 65 per cent. In patients with impairment there was an average audiometric improvement of 3 decibels, which is well within the limits of experimental error. The author concludes that nasal obstruction does not cause impaired hearing and that submucous resection is not to be recommended as a means by which deficiencies in hearing may be improved. [Submucous resection of the septum has often been done in cases of otosclerosis which was mistakenly diagnosed as chronic catarrhal deafness, in the hope of arresting the progression of the deafness, or even of improving the hearing, but with uniformly negative results as far as we know. Johnson's article should help to lay to rest forever this mistaken idea.]

Junemann [30] studied 49 cases by roentgenographic examinations of the temporomandibular joint and by impressions and audiograms; he concludes that the statement that improvement of hearing may be effected by increasing the intermaxillary distance has no scientific proof, and that increasing the intermaxillary distance not only does not improve hearing but fails to relieve tinnitus and pain about the temporal, parietal and masseteric areas in a great majority of cases. He states that roentgenograms of the temporomandibular joint do not show any appreciable difference between cases in which hearing is normal and cases in which it is abnormal. [There is no question that in many cases of nerve deafness or of otosclerosis the bite has been widened to improve the hearing with negative results.

27. Rhegvad, N. R.: Tubar Occlusion or Catarrh of the Middle Ear, Acta oto-laryng. **29**:178 (May) 1941.

28. Van Dishoeck, H. A. E.: Negative Pressure and Loss of Hearing in Tubal Catarrh, Acta oto-laryng. **29**:312 (July) 1941.

29. Johnson, M. R.: Nasal Obstruction and Impairment of Hearing, Arch. Otolaryng. **33**:536 (April) 1941.

30. Junemann, H. R.: Consequences of Shortening the Intermaxillary Distance, J. Am. Dent. A. **28**:1427 (Sept.) 1941.

There do appear to be a very few cases of demonstrable tubal occlusion, with or without secretory otitis, in which the hearing is improved by inflation, the occlusion being due apparently to abnormal closing of the bite, and in these rare instances appropriate dentures are of help.]

Stinson [31] has found Cargile's membrane (sheep mesentery) preferable to cigaret paper for closure of perforations of the drum membrane, since the membrane is moisture resistant and is not irritating. The middle ear must be entirely free from inflammation. If the perforation is of several years' duration, the edges are touched with trichloroacetic acid, after which one or two days is allowed for the inflammatory reaction to subside. The artificial membrane, best made from the thin "fish skin" condom, is cut so that it is at least 25 per cent larger than the perforation. A small, cotton-tipped applicator is moistened and touched to the center of the artificial membrane, which adheres and is easily carried through the speculum to the ear drum, where it leaps into position. If not exactly centered, it can be teased over with a well moistened applicator. In several instances of a very large perforation the anterior portion only was covered at first, and then the remainder as the perforation became smaller.

Healing of the perforation under the membrane requires from ten to thirty days. [Cigaret paper moistened with 1 per cent phenol in glycerin has been more satisfactory in our hands, but we have not been as successful as Stinson appears to have been in obtaining healing under the prosthesis.]

PRIMARY NERVE DEAFNESS

Traumatic Nerve Deafness.—The war is reflected in the increasing number of articles on the effect of acoustic trauma on the hearing.

Perlman [32] suspects acoustic trauma of being the cause of a loss for high tones in any healthy person under 50 years of age without a history or evidence of aural disease, intoxication from drugs, severe constitutional disease or deafness in the family. Loss of hearing from acoustic trauma is not progressive if the patient ceases to be exposed to the stimulus. Tinnitus often accompanies the impairment in hearing and may endure after the acoustic insult. During the active stage of auditory exposure the hearing may fluctuate, depending on whether the examination is carried out immediately after exposure or after a period of rest. The first evidence of acoustic trauma often appears as a localized depression in the region of 4,096 cycles and is usually not noted by the patient. As further trauma occurs, the hearing for speech becomes noticeably affected as the hearing below 4,000 cycles becomes depressed.

Some of the factors which influence the degree of hearing loss from acoustic trauma are: (1) total time of exposure; (2) length of exposure in each period (the ear can recover with rest); (3) loudness of the sound; (4) character of the sound; (5) age (older persons are probably more susceptible); (6) constitutional factors (individual differences in sensitivity); (7) use of protective devices; (8) exposure in a closed space or in the open; (9) previous aural disease, a person with conduction deafness being more resistant to acoustic trauma than a person with normal hearing or one with nerve deafness, and (10) the frequency of the sound.

Clinically Perlman classifies cases of acoustic trauma according to whether the acoustic stimulus was a single explosive sound, a continuous sound or slowly

31. Stinson, W. D.: Closure of Perforations of Membrana Tympani with Cargile's Membrane, Ann. Otol., Rhin. & Laryng. **50**:178 (March) 1941.

32. Perlman, H. B.: Acoustic Trauma in Man, Arch. Otolaryng. **34**:429 (Sept.) 1941.

repeated sound impulses. However, the same type of defect, namely, the dip at 4096 cycles, occurred in all instances.

When normal ears were exposed to a very loud, continuous tone for one hour, the author found that the maximum depression in hearing varied with the frequency of the tone, but was usually one octave above this tone. The dip at 4096 cycles was obtained only when the subjects were exposed to a tone of 4096 vibrations. Sounds of low pitch (64 to 256 vibrations) produced less acoustic trauma than sounds of the same intensity of higher pitch. The frequency of the tinnitus was often an octave above the frequency of the fatiguing tone, and the tinnitus usually stopped before the hearing returned to normal. [These interesting observations suggest that the dip at 4096 cycles, first described as characteristic of acoustic trauma by Shambaugh in 1935, is due either (1) to localized inability of the organ of Corti to recover in the region for 4,000 vibrations or (2) to a greater preponderance of high tones of about 2,000 vibrations in the sounds that result in acoustic trauma.]

Kobrak, Lindsay and Perlman,[33] employing a fatiguing tone of 1024 vibrations at an intensity of 85 decibels for two minutes with normal persons, observed a temporary drop in hearing for this tone, which lasted only two minutes and amounted to 10 to 25 decibels. Since the fatigue was not reflected in the reflex contractions of the tensor tympani muscle when the latter could be observed, the authors regard this fatigue as central in origin. Exposure of the ear of rabbits to a louder tone for forty-five minutes resulted in temporary diminution in the tensor tympani reflex, due to fatigue in the cochlea or in the primary nuclei of the acoustic nerve, more likely in the cochlea. This is the type of fatigue which may result in permanent structural changes if the excessive stimulation continues long enough. A third type of fatigue consisted in a slow relaxation of the tensor tympani muscle during exposure to a continuous tone, and this represents, the authors believe, an acoustic adaptation to the loud tone. The authors point out that the acoustic stimuli of daily life are insufficient to produce any of these types of auditory fatigue. [This paper is a valuable and fundamental contribution to the physiology of the ear and the etiology of traumatic deafness.]

Pastore [34] obtained audiograms on 88 pilots of a commercial air line and found that 42 of them had normal hearing, although their average flying time was four years and 2 had fourteen years of active flying service. The author concludes that pilots are not equally affected by exposure to airplane noises and advises some sort of ear protection for susceptible pilots. [The extreme individual difference in susceptibility to acoustic trauma demonstrated by this author is surprising and significant, for it means that when acoustic exposure in a given industry cannot be decreased, only persons not susceptible to the effects of acoustic trauma should be encouraged to engage in such work.]

Guild [35] reviews the causes of deafness in war, including head injuries, suppurative otitis media, aero-otitis media and acoustic trauma. During 1917 and 1918 the author carried out animal experiments which proved that the most effective of several types of ear protectors in decreasing the damage to the ear from a single detonation, as well as in protecting against prolonged exposure to excessively loud sounds, was a device called the "scientific ear protector," or

33. Kobrak, H. F.; Lindsay, J. R., and Perlman, H. B.: Experimental Observations on the Question of Auditory Fatigue, Laryngoscope **51**:798 (Aug.) 1941.

34. Pastore, P. N.: Hearing Among Experienced Aviators, Proc. Staff Meet., Mayo Clin. **16**:214 (April 2) 1941.

35. Guild, S. R.: War Deafness and Its Prevention, Ann. Otol., Rhin. & Laryng. **50**:70 (March) 1941.

"Tommy," manufactured in England toward the end of World War I. It consisted of a hollow, soft rubber, spherical bulb with an opening on one side, the short opening of the neck being surrounded by a soft rubber flange. The spherical part was 9.5 mm. and the flange 11.5 mm. in diameter, and the wall was 1.5 mm. thick. Proper insertion of the "Tommy" into the external auditory canal was easily accomplished, and it fitted almost all adults without discomfort. The device was easily cleaned and was inexpensive, and there was practically no risk of fragmentation, with formation of secondary projectiles. Voice sounds could be easily heard, but the protector took the "sting" out of intense sounds and effectively stopped detonation waves.

Dickson and Ewing [36] state that deafness for high tones may develop in aviators after as little as one hundred hours of exposure to airplane noise. The first, or warning, sign of such a loss is tinnitus of daily or nightly occurrence following exposure to the noise.

The authors carried out various experiments to determine the character of airplane noise and the most efficient methods of protecting the ears against it. Airplane noise is due chiefly to the engine (exhaust) and the propeller. The highest intensities lie between 30 and 200 vibrations per second; yet the characteristic hearing loss begins at 4096 vibrations. The authors believe the explanation lies in the fact that the transient explosive character of much airplane noise causes stimulation of the whole cochlea (as was shown by Stevens and Davis), and the region responding to 4096 cycles, being anatomically closer to the oval window, is perhaps unduly exposed to such explosive sounds.

The authors tested the efficiency of various types of ear protectors: an ear plug made of rubber and metal; a wax plug covered with cloth, which softens with body temperature so that it can be molded to fit the ear; a tapering closed rubber tube; soft leather ear pads, and a helmet fitted with ear phones for high altitude flying, such as is used under service conditions. They found that far better protection was obtained when the men were trained in the proper use of the protector and that the wax plug gave the highest degree of protection, both for trained and for untrained personnel. When the ear plug was combined with the flying helmet, a still higher degree of protection was obtained. The intelligibility with which speech was heard was found to diminish in proportion to the protection afforded. The authors conclude that partial protection against chronic deafness may be afforded by either ear plugs or a flying helmet, but complete protection requires the use of both. Such a combination is practical for ground personnel but not for aviators, who must be able to hear speech through the ear phones, and who therefore must omit the use of the ear plugs.

Northington [37] describes three types of head injuries which involve the ear:

1. Fractures along the long axis of the petrous pyramid and through the middle ear into the external auditory canal. Blood is present in the middle ear and the external canal, but the inner ear frequently escapes any permanent damage.

2. Fractures through the base of the petrous pyramid anteroposteriorly, with sudden and permanent destruction of vestibular and cochlear function.

3. Concussion with damage to the inner ear, without fracture, and with vestibular symptoms which may be intense. Their intensity, however, is not a criterion by which one may predict the extent of permanent damage.

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36. Dickson, E. D. D., and Ewing, A. W. G.: The Protection of Hearing, J. Laryng. & Otol. **56**:225 (July) 1941.

37. Northington, P.: Functional Ear Damage in Head Injuries, S. Clin. North America **21**:357 (April) 1941.

As soon as possible an accurate record should be made of the presence or absence of blood in the external auditory canal, the hearing, the presence of nystagmus and the intensity of vertigo. The more common type of injury is unilateral partial loss of cochlear and vestibular function. When hearing is present the prognosis is good both for cochlear and for vestibular function.

Nerve Deafness from Drugs.—Falbe-Hansen [38] reports the results of an extensive study of the effects of salicylates and quinine on the ear. Clinical studies showed that a dose of 0.3 Gm. of quinine sulfate produced no effect, but 0.6 to 1.5 Gm. resulted regularly in impaired hearing, tinnitus, a sense of pressure in the ear, vertigo and nystagmus, these symptoms being more severe the larger the dose. Audiograms for 2 subjects receiving 1.5 Gm. of the drug showed a hearing loss for all tones, being somewhat greater for the higher frequencies, but the upper limit for bone conduction, as determined with the monochord, remained normal. As a rule the symptoms disappeared within twenty-four hours. A preexisting hearing defect did not appear to increase the reaction. Two subjects with quinine poisoning, 1 of whom had attempted suicide by taking 7.5 Gm. of quinine sulfate while the other had tried to induce abortion by ingesting 12.5 Gm. of the drug, showed a pronounced reduction of hearing for whisper and the tuning forks, both by air and by bone conduction, with tinnitus, vertigo, spontaneous nystagmus and a sense of pressure in the ears. Within thirty-six hours the hearing was practically normal again and the symptoms were gone. No permanent aural symptoms were observed after the intake of quinine.

Sodium salicylate in a dose of 2 Gm. produced no symptoms, but 3 Gm. or more, given in one dose or fractionally over one to six hours, regularly produced the same aural symptoms as did quinine, with the same results in hearing tests. Normal hearing was completely restored in twenty-four hours. Protracted intake of sodium salicylate, about 5 Gm. daily, produced the same aural symptoms, which disappeared within one to two days after the last dose. Defective ears reacted in the same way as did normal ears, and no permanent effects were noted.

In the second part of his study Falbe-Hansen studied the histologic changes in the guinea pig as a result of quinine and salicylate medication. To minimize agonal or postmortem artefacts, the author used intravital staining. After a single lethal dose of quinine sulfate, or after protracted doses, the organ of Corti showed no distinct structural changes apart from a stronger stainability of certain elements and a sharper definition of the cell borders. In the spiral ganglion the Nissl granules were coarser than normal. Reissner's membrane tended to be depressed over the organ of Corti, often with flattening of the latter, while the secondary tympanic membrane showed evidence of increased perilymph pressure. In the experiment with sodium salicylate the same changes were noted. The author concludes that quinine and salicylate both seem to induce an increased secretion of perilymph, resulting in increased intralabyrinthine pressure, with strain exerted on the fenestrae, producing impairment of hearing and other symptoms. [These observations and conclusions are greatly at variance with the results previously reported by other authors.]

Taylor, Dyrenforth and Pollard [39] carried out lumbar punctures forty-eight hours after birth on 7 infants whose primiparous mothers had received 5 grains (0.325 Gm.) of quinine bisulfate three to four times a day during labor, the total amounts varying from 30 to 120 grains (1.95 to 7.8 Gm.). A total of 7 to 10 cc.

38. Falbe-Hansen, J.: Clinical and Experimental Histological Studies on Effects of Salicylate and Quinine on the Ear, Acta oto-laryng., 1941, supp. 44, p. 1.

39. Taylor, H.; Dyrenforth, L. Y., and Pollard, C. B.: Absorption of Quinine into the Cerebrospinal Fluid of the Fetus in Utero, Ann. Otol., Rhin. & Laryng. **50**:1030 (Dec.) 1941.

of spinal fluid from each infant was tested for the presence of quinine by a technic devised by the authors, and in every case quinine was detected. Examination of the urine of the same infants also showed the presence of quinine in each case. The authors conclude that since it has been proved that quinine is absorbed by the fetus in utero, the drug should be used with caution in the presence of diseases concurrent with pregnancy, and its indiscriminate use as an oxytocic substance may have a malignant influence on the fetal ear. [Another link is supplied in the chain of evidence forged by Taylor that quinine given to the pregnant mother may result in deafness in the child.]

Nerve Deafness from Avitaminosis.—Perlman and Willard [40] contrast the poor clinical results of vitamin therapy in otologic practice with the effects observed in animal experiments, which continue to suggest possible relations between the vitamins and various otologic conditions. Particularly striking were the histopathologic changes in the labyrinthine capsule observed by Mellanby in animals with vitamin A deficiency and confirmed by Loch and Covell. To correlate these changes with the results of qualitative tests of cochlear function, Perlman and Willard studied the acoustic reflex contraction of the tensor tympani muscle in young rabbits before and after they were placed on a diet deficient in vitamin A or its carotenoid precursors. None of the depleted animals became deaf as tested by this response. Histologic examination of their ears showed pronounced hyperplasia of the periosteal layer of the otic capsule on its cranial aspect, as observed by Mellanby, but without the new bone formation in the modiolus or scalae of the cochlea described by that author. The narrowing and lengthening of the internal acoustic meatus produced by the new bone caused compression and stretching of the nerves in the meatus. In spite of this, and in contrast to the observations of Mellanby, no widespread degeneration of the fibers was seen in the sections. However, various degrees of degeneration in some of the cells of the vestibular and cochlear ganglions were observed, apparently due to the stretching and compression of the nerves, with degeneration of the peripheral fibers to part of the basal coil in 1 case.

The authors conclude that the changes observed by them in rabbits were less severe than the changes observed by Mellanby in dogs, probably owing to a difference in the experimental animal. The changes observed in the ear in vitamin A–deficient animals have not been found in human temporal bones. Therefore the information obtained cannot be immediately translated to definite clinical entities, but increased knowledge of the factors influencing bone formation in the otic capsule will ultimately form the basis for a better understanding of the problem of otosclerosis.

Covell [41] emphasizes that although there has been a great mass of experimental work on vitamins, little practical information for the clinician is yet available. The results of experiments in which one vitamin is completely eliminated from the diet of a young animal are not applicable to human beings, who present partial deficiencies of several vitamins; besides, different animals react differently to vitamin deficiencies.

Covell then gives an excellent summary of the experimental observations which have thus far been made on the relation of vitamin deficiencies to the ear. In the otic capsule avitaminosis A results in increase in the periosteal layer sufficient to form exostoses in the internal auditory meatus and the modiolus. Mellanby expressed the opinion that such overgrowth of bone caused pressure on the nerve,

40. Perlman, H. B., and Willard, J.: The Ear in Experimental Vitamin A Deficiency, Ann. Otol., Rhin. & Laryng. **50**:349 (June) 1941.

41. Covell, W. P.: Vitamins and the Ear, Laryngoscope **51**:683 (July) 1941.

with degenerative changes in it. Avitaminosis C results in replacement of the cellular marrow by the fibrous type of tissue present in the periosteal layer. Avitaminosis D causes characteristic lesions in the periosteal and endochondral layers, with replacement of rarefied bone by osteoid tissue. Avitaminosis E causes localized, irregular thickenings of the periosteal layer. Excessive doses of vitamin D in the form of fish oil or irradiated ergosterol resulted in proliferation of periosteal bone in the vicinity of the modiolus. This did not occur if an excess of vitamin A was given with the vitamin D.

In the middle and the internal ear, avitaminosis A results in a relatively high incidence of infections of the middle ear, apparently due to the keratinization of the superficial cells of the mucosal epithelium. The specialized cells of the stria vascularis, the cells of the external sulcus and certain cells in the organ of Corti show degenerative changes, with changes in the cochlear nerve, the spiral ganglion and, to a lesser degree, the vestibular end organ. Avitaminosis B results in infections of the middle ear. Lack of vitamins B_1, B_2 or B_6 causes the most severe changes in the specialized cells of the cochlea and in the nerve. Avitaminosis C results in subperiosteal hemorrhages, fragility of blood vessels and swollen, painful joints. The cavity of the middle ear is filled with a serosanguineous fluid; the submucosa is thickened, and the epithelium is not everywhere intact. The cells of the organ of Corti reveal some swelling, with similar changes in the ampullae. The cochlear nerve, vestibular nerve, spiral ganglion and Scarpa's ganglion show only slight change. Avitaminosis E results in atrophy of the muscles of the middle ear, with low grade infection, which may be responsible for the atrophy.

The author concludes that, except for well established vitamin deficiencies (beriberi, scurvy, pellagra and rickets), there is as yet no positive approach to vitamin therapy, and the actual prescribing of vitamins must be regarded as in an experimental stage. There is evidence in most experimental animals that a correlation exists between diseases of the internal and the middle ear and lack of vitamins in the diet. Since deficiencies in human beings are of a multiple nature, treatment should not be restricted to administration of a single vitamin. [The paper is an excellent summary of present knowledge of the relation of vitamins and the ear.]

Selfridge [42] emphasizes that deafness is caused by all the factors related to growth, not only vitamins but endocrine products, electrolytes and animo acids. If improvement is to result, treatment may have to be followed for years, rather than for weeks or months. The time to begin the prevention of deafness is six months before conception, when both parents should be taught the importance of an optimum diet, which must be continued by the mother through pregnancy and by the child through infancy, childhood and adolescence. Two case reports are presented.

CASE 1.—A girl aged 17 years had been deaf since infancy. Hearing tests showed nerve deafness. In spite of a seemingly normal diet, she was deficient in vitamins A, B and probably C. Administration of a liver and stomach extract with iron in capsules (Lextron) and carotene equivalent to 10,000 U. S. P. units of carotene three times a day, together with a diet rich in vitamin A and 300 mg. of ascorbic acid daily, was instituted. A hearing gain of 10 decibels for the speech frequencies in the right ear and of 8 decibels in the left ear was recorded nine months later. The author suggests that an increased amount of hyperplastic bone may have caused the loss of hearing.

CASE 2.—A girl of 17 had noticed deafness since the age of 8 years. Her basal metabolic rate was —29 and —32 per cent. There was a bilateral defect in conduction hearing, apparently due to adhesions. Aminoacetic acid, thyroid, riboflavin and nicotinic acid, and later the vitamin B complex and vitamin E, were given. A hearing gain of 7 decibels for

42. Selfridge, G.: Present Status of Vitamins in Relation to Eighth Nerve and Conduction Deafness, Arch. Otolaryng. **34**:125 (July) 1941.

speech in the right ear and of 5 decibels in the left ear was recorded seven months later. [These improvements fall within the limits of normal variation, which we take as 10 decibels. The author does not distinguish between the types of deafness that may be due to vitamin deficiencies and the types that have other causes. One gains the impression that he regards vitamins and endocrine imbalance as factors in nearly all cases of deafness. This view is not shared by the authors of the two preceding articles.]

Nerve Deafness from Miscellaneous Causes.—Tamari [43] describes the histopathologic changes in a man of 52 who had had dizziness, tinnitus and impaired hearing for one year and who showed complete deafness on examination. At autopsy gummatous infiltration of the medulla was associated with degeneration of Corti's organ in the cochlea, the result, the author suggests, of destruction of the cochlear nerve fibers in the medulla.

Lurie [44] states that the waltzing guinea pig is a particular strain first described by Ibsen in 1929. The animal tends to run in circles and is deaf, the condition being inherited as a simple mendelian recessive character. There is complete absence of vestibular response to rotation and of vestibular control over movements and positions of the eyes and head and over the righting reflexes, but since histologic examination shows a normal vestibular end organ and vestibular nuclei in every case, it is evident that a central lesion is responsible for the circling. On the basis of experimental work it is concluded that this lesion is probably in the region of the interpeduncular nucleus of the brain stem.

Studies with the Wever-Bray technic show that the deafness in the waltzing guinea pig is complete for all tones above 500 vibrations and for low tones of intensities less than 100 decibels. Histologic examination of the cochlea shows extensive degeneration of the stria vascularis and the hair cells and supporting cells of the organ of Corti. This degeneration seems to begin about at birth, for in embryos examined about ten days before delivery the cochlea was practically normal in all respects while in animals examined five to ten days after birth the apical half of the first turn and the basal half of the second turn of the cochlea showed beginning degeneration. From here the degeneration spreads in both directions, until in older animals only a small portion of the normal organ of Corti remains at the helicotrema. In this degeneration the external hair cells disappear first, then the internal hair cells, then the external pillar cells and, last, the supporting cells, Deiters' cells, the cells of Hensen and, finally, the cells of Claudius, after which the organ of Corti is replaced by a cuboidal epithelium. No clue was found as to how or why the absorption of the organ of Corti is brought about. The same sequence of degeneration, beginning with the external hair cells, was observed in animals made deaf by exposure to loud tones or by poisoning. The spiral ganglion and the cochlear nerve also degenerate, but at a much slower rate. The macula of the saccule does not degenerate, evidence that it is not concerned with hearing.

Ersner and Saltzman [45] present the case of a girl 11 years of age with primary nerve deafness of two years' duration, whose brother and sister had normal hearing. Her mother had bilateral nerve deafness dating from early youth, with 5 normally hearing brothers and 2 normally hearing sisters. The child's grandmother had profound nerve deafness of many years' duration, with 2 normally hearing brothers and 1 deaf sister. The patient, her mother and her grandmother were all more

43. Tamari, M.: Atrophic Degeneration of the Cochlea in Syphilitic Gumma of the Medulla Oblongata, Arch. Otolaryng. **34**:1014 (Nov.) 1941.

44. Lurie, M. H.: The Waltzing (Circling) Guinea Pig, Ann. Otol., Rhin. & Laryng. **50**:113 (March) 1941.

45. Ersner, M. S., and Saltzman, M.: Progressive Analogous Nerve Deafness in Three Successive Generations with Sex-Limited Inheritance, Laryngoscope **51**:241 (March) 1941.

deaf in the right ear. The authors suggest that the deafness of these persons represents a sex-linked hereditary defect of the sensory cells of the cochlea, as a result of which early and progressive degeneration of these cells occurred. That the defect was not bilaterally symmetric, but in each case was worse in the right ear, does not agree with Langenbeck's law of symmetric hereditary deafness. [This interesting report suggests that in this family there may have been a hereditary condition analogous to that described by Lurie in the waltzing guinea pig, but affecting only the cochlea. The possibility of a labyrinthine type of otosclerosis resulting in primary nerve deafness cannot be excluded, however.]

Fowler [46] distinguishes two types of tinnitus: vibratory and nonvibratory. Vibratory tinnitus is mechanical in origin—the result of muscle spasm, vascular bruit or nasopharyngeal squeaks, clicks or the like. Nonvibratory tinnitus is caused by irritation of the auditory nerve elements and is an illusion of sound. The loudness of tinnitus may be estimated by applying a tone of known loudness to the opposite ear and balancing the tinnitus with this tone. It was frequently found by this technic that although a patient complained of intense tinnitus, only 5 or 10 decibels of loudness was required to equal it. The frequency of a tinnitus may be determined by comparison with similar applied frequencies. Tinnitus may be masked in some cases by any frequency and in others by a frequency similar to that of the tinnitus; in still others masking is difficult by any sound. The common tinnitus resembling escaping steam is similar to a thermal noise covering all frequencies to 10,000 vibrations and suggests, therefore, simultaneous irritation of a very large number of neural elements.

The author finds that the presence of tinnitus is always associated with more or less severe deafness, which is due in part at least to the masking effect of the tinnitus itself.

A sure sign of vibratory tinnitus is its audibility to the examiner.

Fowler believes that tinnitus may be of central origin, and he suggests that tinnitus resembling a thermal noise may be of central origin, whereas tinnitus of a particular tone is more likely to be due to localized irritation of the basilar membrane. [The author is to be complimented on his studies of a symptom concerning which so little is known. Particularly puzzling is why a fenestration operation should relieve tinnitus, and why in some cases the tinnitus later returns as loud as before despite an open fenestra and sustained improvement in hearing.]

Treatment of Nerve Deafness.—Katagawa, Shintaro and Yuasa injected an aqueous solution of 0.1 per cent pilocarpine, 5 per cent muscarine, 0.1 per cent acetylcholine, 1 per cent salicylic acid, 1 per cent physostigmine and 1 per cent atropine directly into the middle ear through a perforation or into the retroauricular region in 600 persons with normal hearing, inner ear deafness, chronic suppurative otitis media, deafness due to tubal stenosis and deafness following radical mastoidectomy. In addition, the postauricular region or the meatus was rubbed with a 2 or a 5 per cent preparation of these substances in hydrous wool fat or olive oil. In 43 per cent of subjects a definite, and often marked, increase in the hearing was observed for whisper and tuning forks, the improvement lasting six months. The authors attribute this effect to the pilocarpine, which also caused slight vertigo and nausea on injection into the middle ear or irritation on being rubbed in. Acetylcholine had a similar effect. The senses of smell and taste were

46. Fowler, E. P.: Tinnitus Aurium in the Light of Recent Research, Ann. Otol., Rhin. & Laryng. **50**:139 (March) 1941.

47. Footnote deleted by authors.

noticeably improved also. [Without audiometric substantiation, one cannot accept this bizarre treatment as of actual value in the numerous types of deafness treated by the authors.]

Hofer [48] treated the deafness of old age with androgens and estrogens. Of 20 cases in which these substances were injected intramuscularly, at first every two or three days and later every five or six days, the treatment failed in but 1 case. In the remainder there was noticeable improvement, especially in the tinnitus, lasting one and a half to two years. The author concludes that the glands of internal secretion have a striking effect on the sensory apparatus. [Again, without audiometric proof, one must regard this treatment as of doubtful value.]

The use of prostigmine for impaired hearing was the subject of several contributions, with conflicting conclusions. Rosenthal [49] treated 30 patients with a nasal tampon of 10 per cent mild protein silver for thirty minutes, followed by nasal irrigation and suction and then an intramuscular injection of prostigmine methylsulfate, 1 cc. of a 1:2,000 solution (for children a 1:4,000 solution), three times a week. In no case were there any ill effects. An improvement in hearing of 5 decibels or more was noted in 19 of the 30 cases, and in 16 cases the tinnitus had disappeared or diminished. The pretreatment and post-treatment audiograms are presented. In only 5 cases was there improvement of more than 10 decibels for the speech frequencies. The author concludes that prostigmine has a definite value in treatment of impaired hearing of short duration and of some forms of tinnitus but produces less improvement in chronic deafness. [Of the 5 cases in which there was significant improvement (10 decibels or more for speech), 1 was a case of vertigo, in which hearing frequently fluctuates, 1 a case of impaired hearing of three months' duration following a cold (suggestive of secretory otitis) and another a case of impaired hearing of one week's duration following swimming (suggestive of secretory otitis). In the last case the greatest gain was produced: 16 decibels in one ear and 24 decibels in the other. However, this improvement may reasonably be attributed to the nasal treatments, or it may well have occurred spontaneously. As one of us pointed out recently in evaluating the results of therapy of deafness, one must exclude self-limited conditions, and this the author has failed to do.]

Ersner, Rush and Myers [50] treated 59 patients with prostigmine methylsulfate—10 of whom had conductive deafness, 10 nerve deafness, 14 otosclerosis and 25 mixed deafness. The prostigmine was injected hypodermically, a dose of 1 cc. of a 1:2,000 dilution (0.5 mg.) being given to all patients. The injections were given daily to 25 patients; three times a week to 10 patients; every other day, supplemented with prostigmine bromide tablets (15 mg.) three times a day by mouth, to 14 patients, and three times a week, supplemented with ammonium chloride (enteric) capsules, to 10 patients. Each patient received a minimum of thirty-five injections, except for 3 patients who experienced untoward effects and discontinued the treatment. Audiometric tests showed no objective change in 44 per cent of the patients, who also experienced no subjective improvement; 30 per cent claimed transitory benefit, never substantiated audiometrically; 6.7 per cent claimed improvement without audiometric evidence and experienced the same degree of benefit when a solution of sodium chloride was substituted for the prostigmine, and 13.5 per cent showed audiometric improvement of 8 to 10 decibels,

48. Hofer, G.: Ueber Altersschwerhörigkeit, Med. Klin. **37**:49, 1941.

49. Rosenthal, M.: Use of Prostigmine for Impaired Hearing, Arch. Otolaryng. **34**:540 (Sept.) 1941.

50. Ersner, M.; Rush, I., and Myers, D.: Prostigmine Methylsulfate in the Treatment of Deafness and Tinnitus Aurium, Arch. Otolaryng. **33**:193 (Feb.) 1941.

which proved transitory despite continuance of the therapy. The patients with otosclerosis showed no improvement whatever. The authors conclude that prostigmine is of little, if any, value in the treatment of patients with deafness and tinnitus aurium. [These results are contrary to the observations recorded in the preceding and in the following article but are in accord with our experience; we have never seen impaired hearing or tinnitus benefited by the use of this drug. Unfortunately, there has been widespread newspaper publicity, through the health columns, concerning the beneficial effects of prostigmine on tinnitus and deafness, the claims being based on a few poorly controlled series of cases, without recognition of the more carefully controlled observations of Ersner, Rush and Myers and of Houser, Campbell and Schluederberg,[51] who also concluded that prostigmine is of little, if any, value in the treatment of tinnitus and deafness.]

Alexander [52] regards the use of prostigmine methylsulfate as an advance in the therapy of deafness. the drug being of value in the treatment of acute tubotympanitis, the end stage of acute suppurative and nonsuppurative otitis media, and chronic catarrhal otitis media, with or without tinnitus. Injection of 1 cc. of a 1:2,000 dilution of prostigmine methylsulfate twice a week for three doses was usually sufficient for control of an acute pathologic condition of the tube and middle ear, but a chronic infection may require several months. The other accepted treatments, such as catheterization of the eustachian tube, should be used concurrently. [Since the accepted treatments may be expected by themselves to clear up acute or subacute tubotympanitis, it is difficult to attribute improvement in cases of this condition to the prostigmine.]

Canfield [53] recommends the use of vitamin B complex, in addition to good diet and hygienic measures, in cases of early deafness, in the hope that it may do good when damage to the nerve tissue is not yet irreparable.

Fey [54] cites the fact that deaf-mutes suffer less from a depressive state of mind than do persons deafened later in life as evidence that this depression is the result less of the actual disability than of a psychic traumatic neurosis, due to the subjective feeling of one's being imperfect. Such a reaction can be overcome by psychic effort, exercised spontaneously or through psychotherapeutic guidance and influence.

The author calls attention to the fact that whereas the blind are objects of pity in fiction, the deaf are usually objects of fun—evidence that the world as a whole does not regard deafness as seriously as do the deafened themselves and proof of the highly exaggerated opinion of their handicap possessed by the deafened. [It is almost impossible for persons with normal hearing to appreciate what it means to be cut off from one's fellows by deafness. Only when one sees the psychologic transformation that occurs when the hearing is partially restored in a case of chronic deafness does one realize the degree to which the deafened person is cut off from ordinary, everyday conversation. Solitary confinement has long been regarded as a special form of severe punishment. The deafened live in partial solitary confinement. The societies for the hard of hearing are doing wonderful work in helping these persons to find companionship and social adjustment.]

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51. Houser, K. M.; Campbell, E. H., and Schluederberg, H.: Treatment of Chronic Deafness and Tinnitus Aurium with Prostigmine, J. A. M. A. **115**:994 (Sept. 21) 1940.

52. Alexander, L. W.: The Use of Prostigmin Methylsulfate in Deafness: A Preliminary Report, New Orleans M. & S. J. **93**:474 (March) 1941.

53. Canfield, N.: Therapy of Deafness, Internat. Clin. **1**:192 (March) 1941.

54. Fey, H.: Psychological Aspects of Deafness, J. Laryng. & Otol. **56**:81 (March) 1941.

Ménière's Syndrome.—Furstenberg, Richardson and Lathrop [55] have treated more than 150 patients suffering from Ménière's disease with a salt-free diet and ammonium chloride. In a follow-up examination of 35 of these persons, all but 6 of whom had been treated for more than a year, the authors found that 57 per cent had been free from vertigo, 26 per cent were improved and 17 per cent were unimproved, and they concluded that the results of treatment were unsatisfactory. Tinnitus was relieved in 3 per cent, diminished in 48 per cent, unchanged in 40 per cent and increased in 9 per cent. Hearing was improved in 9 per cent, unchanged in 65 per cent and further impaired in 26 per cent.

Of the 17 per cent whose vertigo was not relieved, 2 were unable to tolerate the ammonium chloride and refused to conform to the diet; 1 improved until a heart block developed, for which he was given theophylline with sodium acetate; 1, a traveling man, was unable to adhere to the diet, and for 2 patients treatment resulted in unexplained failure.

The authors emphasize the importance of hospitalization during the first week the patient is receiving the diet in order that he may familiarize himself with the theory and practice of the strict regimen and may avoid taking self-prescribed nostrums containing sodium. It is important that the water supply should not contain sodium chloride.

Ammonium chloride is most effective in elimination of sodium from the diet if given for three days and then omitted for two, and it should be administered in 7½ grain (0.488 Gm.) capsules, 6 with each meal, taken between bites of food, three times a day. Enteric-coated capsules are unsuitable, since they may be eliminated unchanged.

The authors recommend strict observance of the full regimen for six weeks and then discontinuance of the ammonium chloride for six weeks; then, if the patient is symptom free, he may eat food cooked with salt, provided it is not highly seasoned with salt and he never adds salt at the table. A recurrence of symptoms requires immediate reversion to the original program.

The authors conclude that Ménière's disease appears to be due to or associated with an accumulation of sodium ions in the labyrinth, producing a pressure or chemical alteration which causes the attacks of vertigo. Dietary management will relieve the vertigo in the majority of cases but does not affect the tinnitus or the hearing. Surgical intervention should be resorted to only when an adequate trial of medical therapy is fruitless.

Grove [56] believes that allergy, avitaminosis and disturbances in the endocrine balance must be given more serious consideration in cases of Ménière's syndrome. The waterlogging that evidently occurs in the labyrinth may also involve the central cochlear nuclei, in view of the fact that tinnitus may persist after destruction of the labyrinth or section of the nerve. The success of medical treatment by dehydration (Mygind and Dedering), elimination of sodium (Furstenberg) or administration of histamine (Shelton and Horton) indicates that surgical intervention should be reserved for patients who have not responded to a medical regimen.

Atkinson [57] believes that Ménière's syndrome is of vasomotor origin, the result either of allergy (vasodilatation) or of vasospasm. Allergy as a cause of Ménière's

55. Furstenberg, A. C.; Richardson, G., and Lathrop, F. D.: Ménière's Disease: Addenda to Medical Therapy, Arch. Otolaryng. **34**:1083 (Dec.) 1941.

56. Grove, W. E.: An Evaluation of the Ménière Syndrome, Ann. Otol., Rhin. & Laryng. **50**:15 (March) 1941.

57. Atkinson, M.: Observations on the Etiology and Treatment of Ménière's Syndrome, J. A. M. A. **116**:1753 (April 19) 1941.

syndrome in some cases has been established, but the discovery of the specific allergen may be difficult and tedious. The author has found that the allergic patients are histamine sensitive, as shown by the intradermal injection of 0.0057 mg. of histamine base (0.01 mg. of histamine dihydrochloride). In the sensitive subject a large wheal (½ to ¾ inch [1.3 to 1.9 cm.]) appears, with long trailing pseudopods 1 inch (2.5 cm.) or more in length forming in three to five minutes; these begin to fade only after twenty minutes and are still apparent at the end of thirty minutes. The appearance of small buds at the edge of the main portion of the wheal in ten to twenty minutes is not assessed as a positive reaction. In doubtful cases 0.02 mg. of histamine dihydrochloride results in a decided increase in reaction in sensitive persons, but not in insensitive ones.

In 200 histamine cutaneous tests no patient who was undoubtedly sensitive failed to respond to histamine desensitization. Seven histamine-sensitive patients were entirely relieved of their attacks for many months. Doubtfully sensitive or insensitive patients not only failed to respond but sometimes were made worse. Of the 7 who were sensitive, 5 had other allergic manifestations, all of which were relieved by treatment with histamine. Several of these persons experienced a Ménière attack after an injection of histamine, a response suggesting that this group of patients is unduly sensitive to the drug, the Ménière attacks being due to a local reaction in the labyrinth similar to the cutaneous reaction.

This allergic, or histamine-sensitive, group, however, comprises only the minority of patients with classic attacks of Ménière's syndrome. The larger group (19 patients in the author's series) present a negative response to histamine, and their attacks, it is postulated, are due to vasospasm. They are often older patients, who can be treated with vitamin B_1, a vasodilator, with benefit. Amyl nitrite pearls in mild doses gave relief from an attack in 2 cases. Many of this second group have migraine, and the vasodilator acetylcholine, 125 mg., injected subcutaneously during the height of an attack, gave immediate and complete relief. Amphetamine sulfate, 5 mg., by mouth, produced a severe attack thirty-five minutes later in 1 case but on other occasions on which it was followed by an injection of nicotinic acid, a powerful vasodilator, no attacks occurred. Epinephine hydrochloride, 1 cc. of a 1:1,000 dilution, injected subcutaneously, produced nystagmus, past pointing and sway toward the side of the affected ear in all of 5 cases, but without an attack; nicotinic acid, given intramuscularly, relieved these symptoms.

The author suggests that the beneficial effects of histamine during an attack, as reported by Horton, are due to its vasodilating effect, and not to any specific action. The final proof of the theory of vasoconstriction is the therapeutic response, and 17 of 19 patients treated with vasodilator substances were completely relieved or showed steady improvement, while 2 showed initial improvement but had an eventual relapse.

The histologic changes in Ménière's syndrome—gross distention of the endolymphatic system with degenerative changes in the sensory elements—may be explained as the result either of allergy or of local vasoconstriction, followed later by vasodilatation and increased capillary permeability.

In the treatment of Ménière's syndrome the two etiologic varieties must first be differentiated by the intracutaneous histamine test and the clinical characteristics. Patients with the allergic type are younger; the hearing loss is not great nor the tinnitus severe; the attack is preceded by headache, greater impairment of hearing and tinnitus, and after the attack all of these symptoms improve. If possible, the allergen is discovered and removed. If this measure is unsuccessful, histamine desensitization is started with 0.01 mg. given subcutaneously, the dose

being gradually increased to the limit of tolerance, usually 1 mg. or less, in injections two or three times a week; the maximum dose is then continued once a week for a month. After three months the course of injections is repeated. Headache, vertigo and other allergic symptoms are relieved, but deafness and tinnitus are not helped so often.

Patients with the vasospastic type are generally older, but a few are young and usually experience severe progressive symptoms, as a rule bilateral and resistant to treatment. In the middle-aged the attacks come in groups, and the general course is toward deterioration, while in the aged the symptoms are mild. As a rule the deafness and tinnitus are more severe in the vasospastic than in the allergic group; the attacks of vertigo come without warning and are followed by increased deafness and tinnitus, with persistent unsteadiness for several days. The vitamin B complex, especially the nicotinic acid fraction, is effective with the young and middle-aged in the vasospastic group, while the sedative effect of thiamine hydrochloride is more important with the elderly patients. The dose of nicotinic acid is 50 mg. or more daily, given intramuscularly, for the middle-aged patients and 10 to 25 mg. daily for the young and old patients. The daily dose of thiamine hydrochloride is 5 mg. for the younger and 10 to 25 mg. for the older group. Parenteral treatment is advised at first and oral administration later.

Histamine will make worse the group with the vasospastic type and nicotinic acid the group with the allergic type—hence the importance of accurate differentiation.

Treatment by limitation of fluid and of the sodium-potassium intake has been disappointing in the author's experience. Stricture of the eustachian tube is frequent and should be treated energetically with bougies. The infective theory is viewed with great skepticism. [The author's speculations on the pathogenesis of Ménière's syndrome are novel and interesting, but they seem to oversimplify the problem. We agree that food allergy is one of the causes of this baffling condition, but stricture of the eustachian tube has not been present in any of our cases.]

Atkinson [58] regards the normal ratio of the time of onset of nystagmus in the cold caloric test to its duration as 1:4. With the hyperactive labyrinth the ratio is increased to as much as to 1:7; with the hypoactive labyrinth it is diminished to 1:2, or even to 2:1. In patients with the allergic type of Ménière's syndrome the vestibular response is unchanged or is diminished, owing to an excess of endolymph, with damping down of the hair cells in the semicircular canals. In persons with the vasospastic type the vestibular response is invariably increased, owing to diminished blood supply and increased irritability of the nerve endings. However, in cases of long-standing vasospasm there may be gradual degeneration, with hypofunction replacing the hyperfunction.

Horton [59] believes that the ultimate treatment of Ménière's disease will be medical, not surgical. For the acute attack he administers 1.88 mg. of histamine phosphate or 2.75 mg. of histamine diphosphate (1 mg. of histamine base) in 250 cc. of physiologic solution of sodium chloride intravenously at the rate of 50 to 60 drops per minute by gravity over one and a half hours. If a single dose does not relieve the symptoms, it may be repeated daily.

For the prevention of future attacks histamine phosphate, 0.1 to 0.2 mg. subcutaneously, two to four times a week, is then given as a maintenance dose.

58. Atkinson, M.: Changes in Vestibular Sensitivity in Ménière's Syndrome and Their Significance, Arch. Otolaryng. **33**:969 (June) 1941.

59. Horton, B. T.: The Use of Histamine in Ménière's Disease, Surg., Gynec. & Obst. **72**:417 (Feb.) 1941.

Of 49 patients treated with histamine, 15 were known to be well; the others had had one or more recurrences, possibly because they were not taking the maintenance dose of histamine.

The author suggests that Ménière's syndrome is a local process involving the labyrinth, most likely an altered permeability of the capillary walls, resulting in local edema, and that histamine acts on the permeability of the capillaries. He does not believe that loss of hearing is necessary for the diagnosis. The average caloric response was normal or slightly hyperactive.

Hutchinson [60] describes an apparatus for injecting alcohol into the labyrinth through the intact drum membrane and the footplate of the stapes in cases of intractable and extreme aural vertigo. The apparatus consists of an electric otoscope with a magnifying lens attached to an 8 inch (20.3 cm.) rod and marked with three hair lines, one a prolongation of the axis of the rod and each of the other two at an angle of 20 degrees with it. An aural speculum for the otoscope with a lateral slot, a syringe marked in minims and a stiff, angled, short-beveled needle complete the apparatus. With the patient under light general anesthesia and the speculum in the external auditory meatus, the lens is rotated until the rod cuts the lower edge of the orbit on the side of operation, the apparatus being thus set at Reid's base line. Slight movements of the operator's head bring the middle hair line to pass through the umbo and the upper hair line through the tip of the short process. The needle is now introduced at a point on the upper hair line close behind the short process. This will impinge on the foot plate of the stapes. Further light pressure then suffices to drive the needle through the foot plate into the labyrinth, the sensation of perforating the bone being unmistakable. If there is no twitching of the angle of the mouth, 1 minim (0.062 cc.) of alcohol is introduced and the needle is withdrawn. [If this method proves effective and safe, it should be preferable to craniotomy and section of the nerve in cases of intractable Ménière's disease.]

122 South Michigan Boulevard.
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60. Hutchinson, C. A.: Aural Vertigo: Alcohol Injection of the Labyrinth, J. Laryng. & Otol. **56**:439 (Dec.) 1941.

Abstracts from Current Literature

Ear

TYMPANIC CAVITY IRRIGATION. J. KENT LEASURE, Ann. Otol., Rhin. & Laryng. **51**:253 (March) 1942.

Leasure's method of treatment in certain cases of otitis media consists of forcing a thin fluid from the external auditory canal through a perforation in the tympanic membrane, into the tympanic cavity and on down the eustachian tube into the nasopharynx. The solution should be warmed, the patient's head tilted to the opposite side and the fluid allowed to fill about one third of the canal. Pressure is exerted by means of a Siegle otoscope, and the fluid will disappear; at the same time bubbling will be noted at the nasopharyngeal orifice of the tube. Thorough cleansing of the external canal previous to the treatment is essential, and the method should not be attempted if polypi are present. If there is a question of the patency of the tube, air should be used first, before fluid is tried. The author feels that this method is of use in cases in which a long-standing perforation is present, so that each new infection is allowed to creep up the eustachian tube and involve the tympanic cavity. He has also used the treatment in cases of the acute condition, without development of mastoiditis. Roentgenographic studies have been made before, during and after treatment, and no ill effects of the treatment have been noted. The nasopharynx must be treated along with the ear. The solution should be aqueous, and the drug is selected according to the organism present.

M. V. MILLER, Philadelphia.

HISTAMINE IN THE TREATMENT OF MÉNIÈRE'S SYNDROME. MILES ATKINSON, J. A. M. A. **119**:4 (May 2) 1942.

Atkinson discusses Ménière's syndrome and reviews the two types, namely, that in which sensitivity to histamine is proved by cutaneous tests and that in which sensitivity to histamine does not exist.

Ten patients were worse after injections of histamine, 3 showed no change and 2 improved, though nicotinic acid was necessary to maintain the improvement.

The author concludes that histamine therapy is suitable for selected patients only and that a patient should be assigned to his correct group before effective treatment can be assured.

He emphasizes that histamine possesses two effects: the immediate, a dilator action, and the remote, resistance to this action, much the same as the resistance produced by a vaccine to the effects of a micro-organism—often a vasoconstrictor effect.

The repeated administration of histamine, though effective as a desensitizing agent with patients showing excessive vasodilation, fails with the group suffering from vasoconstriction, and often makes matters worse.

It is interesting to note that nicotinic acid was generally far more valuable than histamine, helping to maintain the improvement in the 2 cases already mentioned, as well as in almost all of the others.

GORDON, Philadelphia.

OTOSCLEROSIS: RESULTS WITH THE FENESTRATION OPERATION. GEORGE E. SHAMBAUGH JR., J. A. M. A. **119**:243 (May 16) 1942.

The author discusses the history, technic, indications for operation, evaluation of results and complications of the fenestration operation and concludes with a definite statement of principles the important points that he has noted during a careful observation of three and a half years.

The article is well illustrated. Diagrams outline the important procedures. The operating setup is illustrated. Many audiograms with analyses are shown.

The author has given full credit to all the otologists of Europe and America who have pioneered and created the various steps that have brought this operation to the present state of near perfection and adds his own technic of constant irrigation "to wash away every particle of bone dust while making the window in the bony horizontal semicircular canal." He also introduces the use of the binocular dissecting microscope "to aid in the visualization of the very tiny structures while making the window."

Summary.—1. The fenestration operation offers the possibility of partial restoration of impaired hearing, as a result of fixation of the stapes. 2. The problem of closure of the window has been solved by the technic which he created. 3. The improvement in hearing, as seen in the audiograms, is gratifying, and the results are better than those obtained with a hearing aid. 4. Occasionally poor results are obtained; hence the poorer hearing ear should be selected for operation. 5. An improvement in hearing which is maintained for more than six months is probably permanent. 6. Properly done, the fenestration operation carries little risk to life, health or useful hearing.

GORDON, Philadelphia.

SULFANILAMIDE IN OTITIS MEDIA IN CHILDREN: FURTHER REPORT ON A CONTROLLED SERIES. EDWIN B. BILCHICK and GEORGE HUNTER O'KANE, Laryngoscope **52**:341 (May) 1942.

Bilchick and O'Kane compare the results in their two controlled series of children with otitis media who were treated with sulfanilamide or a derivative. The first 103 patients were treated during January, February and March 1939, 55 being given sulfanilamide and 48 being used as controls, without any sulfonamide drug. During the winter months of 1940, under the same conditions as those observed the year before, 80 patients were studied, one half receiving a sulfonamide drug and the others none. A different dose of the drug was used in the two series. In 1939 moderate doses were employed, while in 1940 larger doses were given earlier in the disease. In the 1940 series, 200 mg. of sulfanilamide or an allied compound per kilogram of body weight was given in divided doses during the first twenty-four hours, followed by a daily amount of 120 mg. per kilogram. Sodium bicarbonate was administered with the drug. The medication was continued for from four to seven days after apparent cure. In the 1939 series, who received moderate doses of the drug, the duration of the aural discharge for the sulfanilamide-treated patients was one-half that for the controls, but the number of mastoidectomies necessary was not affected. In the 1940 series, the use of the sulfonamide drug earlier and in larger doses reduced the duration of the disease one third and the number of mastoidectomies over one half. No serious complications resulted from the therapy. There were 5 cases of early and 3 cases of late recurrence of the otitic infection between one and six weeks after the ear was dry and the drug was stopped. In several of these cases repeated myringotomy was necessary. There were no relapses in the control series.

WOOD, Newark, N. J.

INDUSTRIAL OTOLOGY IN CAISSON WORKERS. RALPH ALMOUR, New York State J. Med. **42**:779 (April 15) 1942.

Almour recites the history of aural disturbances among compressed air workers, reports cases of his own and gives his conclusions based on a study of ten years. He recognizes three types of auditory and tympanic disability, which may be briefly headed as mild, moderate and severe, and summarizes his study thus: The clinical picture of blocked ear is an entity, a problem for the otologist, which will become greater with increase in air travel. Intensive study of the problem is imperative, and labor unions should be more acutely aware of its implications. There should be a disinterested board of examiners who should make studies in individual cases of blocked ear, and otologists should establish a standard by which degrees of disability may be judged, so that legal and controversial elements may be reduced to their lowest terms. Otologists should set up a standard of "compensability" for all loss of hearing from trauma and eventually should extend this to cover all impairment due to injury. Otologists should be careful not to contradict each other in court or out, for so many factors enter into a case set for judgment as to make fair conclusions difficult. Almour thinks that blocked ear is so seldom permanent and debilitating as to make "compensable loss in hearing" infrequent. The hearing of all applicants for "sand hog duty" should be carefully measured before they are put to work. One might then have a standard with which to compare any subsequent loss of hearing. Too rapid decompression is the chief cause of blocked ear in caisson workers, tubal closure being the concomitant factor.

VOORHEES, New York.

Pharynx

A PRELIMINARY REPORT ON THE TREATMENT OF AGRANULOCYTOSIS WITH SULFATHIAZOLE. W. DAMESHEK and L. E. WOLFSON, Am. J. M. Sc. **203**:819 (June) 1942.

Despite the claims made for the pentose nucleotides, adenine sulfate, liver extract, transfusions, yellow bone marrow extract, and so on, the treatment of agranulocytosis has failed to change the essentially fatal outcome of the disease. Death is probably the result of overwhelming sepsis in a body stripped of its granulocyte defenses. In order to combat sepsis, sulfathiazole

(2-[paraaminobenzenesulfonamido]-thiazole) in large doses was given in addition to blood transfusions and pentnucleotides in 2 cases. In both cases recovery followed. Although no proof is present that the sulfonamide drug was curative, it did no harm, and the authors believe it may have been of value in combating the secondary sepsis. Further trials in other cases of agranulocytosis are indicated. Apparently, the sulfonamide compounds may have their characteristic action in the absence of granulocytes. In 1 of the cases reported the total leukocyte count was 850 and in the other 500. In neither case were any granulocytes present in the circulating blood, and sternal puncture showed almost complete absence of granulocytes. The ensuing recoveries may have been due, in part at least, to the action of the sulfonamide drug on the sepsis which was almost certainly present, an effect allowing spontaneous leukocytic regeneration in the bone marrow to take place.

DAVISON, Danville, Pa.

TREATMENT, COMPLICATIONS AND DEATHS IN SEVEN HUNDRED AND FIFTY-THREE CASES OF CLINICAL DIPHTHERIA. Y. TOGASAKI, L. ROSOVE, A. G. BOWER and P. M. HAMILTON, Am. J. M. Sc. **204**:218 (Aug.) 1942.

The authors discuss the observations on and treatment of 753 patients with diphtheria who were admitted to the Los Angeles County Hospital between July 1, 1937 and June 30, 1940. The total mortality rate was 4.8 per cent. There was a mortality of 100 per cent among 9 patients with the "bull neck" type, in which the membrane extended into the larynx, trachea and bronchi. The dose of antitoxin varied between 20,000 and 40,000 U. S. P. units and was determined by the apparent toxemia, the extent of the membrane and the duration of the illness. The mortality rate was lowest in those patients who received antitoxin before the third day of illness. Vitamin B complex and vitamin C were given in large doses. Circulatory failure, the result of myocarditis, was one of the most frequent complications. Dextrose and insulin were given intravenously to combat early circulatory failure. For treatment of late circulatory failure, 0.25 to 1 cc. of pitressin was given subcutaneously every eight hours, or oftener if necessary, in order to bring the blood pressure up to normal. Adrenal cortex extract in doses of 10 to 20 cc. was also used in some cases to maintain blood pressure. In most cases in which relief of laryngeal obstruction was required, tracheotomy was more satisfactory than intubation.

DAVISON, Danville, Pa.

Nose

ESTROGENIC SUBSTANCE IN THE TREATMENT OF ATROPHIC RHINITIS. LOUIS A. SAFER, Ann. Otol., Rhin. & Laryng. **51**:158 (March) 1942.

The author reports the result of treatment in 20 cases of atrophic rhinitis with estrogenic substance for a period of one and one-half years. The nose is first cleansed of all crusts and then packed with large cotton pledgets moistened with estrogen. These are placed in the region of the middle meatus, with strings attached to the pledgets so that the patient can draw them out. The packs are to be allowed to remain in place as long as possible; at first they are tolerated only a few hours, but later they may remain for several days. Treatment is given twice weekly. After the pack is withdrawn the patient is instructed to irrigate his nose with a warm alkaline solution each day until the next treatment. In addition, 0.25 cc. of the estrogenic substance is sprayed into each nostril twice a day. After improvement is noted the irrigation with the alkaline solution is stopped and the estrogenic substance is sprayed into the nose only once a day. The results obtained in these cases were excellent, but the author feels that the therapy is palliative, and not curative; nevertheless, it is a highly useful addition to the therapeutic armamentarium.

M. V. MILLER, Philadelphia.

THE RESULTS OF TREATMENT WITH BACILLUS COLI METABOLIN IN ALLERGIC RHINITIS. HARRY H. SHILKRET, New York State J. Med. **42**:332 (Feb. 15) 1942.

Shilkret makes an exhaustive analysis of the work of Elsbach, concerned with a "cure" for hay fever (*N. Y. State J. Med.* **41**:1248 [June 15] 1941). In that article Elsbach reported 75 cases of "desensitization" of the nasal mucous membrane with a preparation containing the products of metabolism of Bacillus coli found in the human intestine (B. coli metabolin). Newspapers played up the "discovery," and many physicians throughout the United States asked for supplies of the agent so that they might try it on their patients. In the City Hospital and in the French Hospital Shilkret studied 20 cases intensively. He tabulates his protocols and his results. In order that no other investigator may travel over the same road and follow an *ignis fatuus*, Shilkret concludes: "B coli metabolin is of no value in the treatment of hay fever or allergic coryzas."

VOORHEES, New York.

A RÉSUMÉ OF CONSERVATIVE SINUS MANAGEMENT. J. HALLOCK MOORE, South. M. J. **34**: 848 (Aug.) 1941.

The author states that the most common etiologic factor in disease of the sinuses is nasal obstruction and adds that any impairment of the nasal airway must be corrected before normal physiology of the sinus is reestablished.

In the conservative management of sinus disease, shrinkage of the nasal mucous membranes, stimulation of the patient's resistance by the use of vaccines and chemotherapy, drainage by any method, careful irrigations with physiologic solution of sodium chloride, protein silver packs followed by irrigation, heat in the form of infra-red light and diathermy and roentgen therapy are advised. The use of irritants in the nose and mass suction above 180 mm. of mercury should be avoided.

In cases of acute sinusitis with pain and fever the patient is put to bed, and the discomfort is controlled with morphine and codeine. When the antrum is acutely involved irrigations are started as early as the second or third day and repeated daily, or less frequently as indicated. The antrum is entered through the middle meatus and is washed with physiologic solution of sodium chloride.

Chronic infections of the antrum are treated with irrigations of saline solution, vaccines and chemotherapy for four to six weeks before surgical therapy is resorted to. Permanent nasoantral openings will usually suffice in cases in which the more conservative measures have failed.

SINGLETON, Dallas, Texas.

Miscellaneous

COMPLETE CICATRICIAL STENOSIS OF THE ESOPHAGUS: PERMEATION MADE POSSIBLE BY EXTERNAL OPERATION IN CERTAIN CASES. FLETCHER D. WOODWARD, Ann. Otol., Rhin. & Laryng. **51**:94 (March) 1942.

Woodward points out that of the cases of accidental injury, those in which the lesion was due to ingestion of a caustic through error, the stricture was in the upper third of the esophagus in 53 per cent, in the middle third in 24 per cent and in the lower third in 24 per cent. Of the cases in which the lesion developed after attempted suicide, the stenosis occurred in the middle third in 71 per cent and in the upper and lower thirds in 11 per cent each. This, he says, is due to the deliberate swallowing of a larger amount of the caustic. The upper third is most accessible to the external surgical approach, but any part may be approached by modern surgical means.

The emergency treatment of the acute condition is prompt lavage of the face, mouth, esophagus and stomach with the proper neutralizing solution. A rubber feeding tube is inserted through the nose to the stomach and left in place for from ten to fourteen days. Roentgenographic or esophagoscopic studies are not made for three weeks. If there is beginning stricture, careful and gentle bouginage is carried out through the esophagoscope.

Most patients appear from two to four months after ingestion of the caustic, when they begin to have difficulty in swallowing, with pain and loss of weight. They are studied after ingestion of an opaque meal and then with an esophagoscope. If the stricture is not too severe, dilatation is carried out with the Jackson flexible bougies under visualization. If the stricture is pronounced, the patient is referred to the surgeon for a two stage gastrostomy, as the sooner the patient can receive proper food and liquids the earlier the more active treatment can be started. Gastrostomy should be done with due consideration to its type and location for future retrograde bouginage. When the gastrostomy wound is healed, an attempt is made to have the patient swallow a string. The free end is then fished out of the stomach and tied to the upper end on the outside. When the string is in place, future treatment is carried out by the retrograde method of Tucker. Usually treatment is necessary for about two years before the gastric opening may be closed, and even after that dilatation may be necessary.

If these measures fail, the patient must keep the gastrostoma for life, run the risk of perforation by bouginage or undergo surgical treatment. The external route for the operation is advocated, and cases are reported, with the method used in each.

M. V. MILLER, Philadelphia.

ORBITAL CELLULITIS. L. J. WALLNER, Ann. Otol., Rhin. & Laryng. **51**:127 (March) 1942.

Orbital cellulitis is an acute inflammation of the soft tissues of the orbit, characterized by redness and edema of the lids, proptosis, limitation of the globe, chemosis of the conjunctiva and a septic temperature. The veins are important. The superior and inferior ophthalmic

veins drain the area supplied by the ophthalmic artery; the superior ophthalmic vein receives the anterior and posterior ethmoid veins and has a large communication with the facial system through the angular vein; the inferior ophthalmic vein communicates with the pterygoid plexus; the two ophthalmic veins do not follow the artery out of the orbit but leave through the superior orbital fissure to enter the anterior wall of the cavernous sinus. There are no lymphatics or nodes in the orbit. In 70 to 80 per cent of cases the condition follows acute sinusitis—frontal sinusitis in adults and ethmoiditis in children. Cases are much more frequent in infants and young children than in adults. Trauma from penetrating wounds or foreign bodies in the orbit may be the cause. The pathologic change may be a true cellulitis of the soft tissues, progressing to abscess, a subperiosteal abscess between the periosteum and the bone of the orbit or thrombophlebitis of the orbital veins. The author believes that venous stasis is important in producing the clinical picture and cites experiments to indicate this. Differentiation of orbital cellulitis and thrombosis of the cavernous sinus may be difficult or impossible. Conservative treatment will be sufficient in many cases of cellulitis.

M. V. MILLER, Philadelphia.

SEASONAL AND GEOGRAPHIC INFLUENCES ON FOOD ALLERGY. A. H. ROWE, J. Allergy **13**:55 (Nov.) 1941.

The author calls attention to the relief of bronchial asthma, allergic bronchitis and nasal allergy in food-sensitive persons when they travel from the ocean or bay shores over the first range of hills in northern California. Suggested causes of this maritime effect on food allergy, especially when bronchial asthma or nasal allergy is involved, include humidity and the mineral content of the ocean air. The effect is doubtless due to the so-called secondary causes of allergy, which include odors, smoke, weather, changes in temperature, nervousness and fatigue.

Food allergy in certain patients, especially when it produces bronchial asthma, nasal allergy and atopic dermatitis, becomes less active, or even absent, in the summer months, increasing its activity to a maximum during December and January.

Not all food-sensitive patients are affected by these factors, those with bronchial and nasal allergy apparently being most susceptible.

The author states that no symptoms due to inhalant allergy formed the basis of his conclusions. The various discussants of the paper expressed the belief that the inhalant factors required consideration.

FRIEDBERG, Chicago.

INFLUENZAL MENINGITIS: THE SUCCESSFUL TREATMENT OF A CASE IN AN INFANT. MARGARET STRANGE BRYAN and JOHN H. MUSSER, New Orleans M. & S. J. **94**:188 (Oct.) 1941.

The authors present a clinical report of a case of influenzal meningitis with recovery in an infant of 11 months. The treatment included use of sulfapyridine, influenza serum and transfusions. As often happens in cases of recovery from influenzal meningitis, the multiplicity of therapeutic agents used makes it impossible to evaluate the curative effect of any of them.

BERKLEY, Los Angeles. [AM. J. DIS. CHILD.]

THE KNOTT TECHNIQUE OF ULTRAVIOLET BLOOD IRRADIATION IN ACUTE PYOGENIC INFECTIONS: A STUDY OF 103 CASES WITH CLINICAL OBSERVATIONS ON THE EFFECTS OF A NEW THERAPEUTIC AGENT. GEORGE MILEY, New York State J. Med. **42**:38 (Jan. 1) 1942.

Miley states that the detoxification effect of ultraviolet radiation is not generally known, in spite of the appearance of many articles in the literature since Knott and Hancock (*Northwest Med.* **33**:200 [June] 1934) reported complete recovery following ultraviolet irradiation of the blood in 1 case of general septicemia and in 1 case of brain abscess. Miley cites 41 articles from the literature. A definite amount of blood is drawn from a patient's vein, irradiated with ultraviolet rays and returned by the same route to the circulation. To do this an irradiation chamber, an automatic transfusion pump and a water-cooled mercury-quartz burner must be at hand. The amount withdrawn is calculated according to a given formula and rarely exceeds 300 cc. It is stirred with 2.5 per cent sodium citrate, in a proportion of 1 part citrate to 5 parts of blood. This mixture is poured through sterile gauze into a transfusion chamber and then "pumped" back into the patient's vein. A typical case was that of a woman aged 31 with hemolytic streptococcus oropharyngitis, diagnosed by culture. The temperature on admission was 102.1 F. Three hours after irradiation it was 101.2 F. On the third day after the first intravenous treatment only a few "mildly hemolytic anaerobic strepto-

cocci" were present. The patient was discharged five days later as recovered. However, in cases of infections due to Staphylococcus aureus results were not so favorable, but in over 1,000 infusions no harmful effects were seen and in no case in which this irradiation method failed did chemotherapy succeed. Miley states that this procedure offers better hope of recovery from pyogenic infections than any other therapy yet known. VOORHEES, New York.

TREATMENT OF FRESH TRAUMATIC AND CONTAMINATED SURGICAL WOUNDS. J. D. BISGARD and C. P. BAKER, Surg., Gynec. & Obst. **74**:20 (Jan.) 1942.

As a result of their experimental and clinical experiences, Bisgard and Baker condemn the use of bactericidal agents in contaminated wounds. Moreover, they report that in rabbits bactericidal agents caused moderate to severe damage of tissue and interfered with bone repair either by direct action or through failure to prevent infection.

Sulfanilamide, which apparently did not damage tissue and did not interfere with bone repair, was more effective than antiseptics in preventing suppuration of contaminated wounds.

The essayists believe that infection in contaminated fresh traumatic and operative wounds is most effectively prevented by copious mechanical washing with saline solution followed by débridement and the implantation of sulfanilamide powder. These measures, of course, follow rapid preliminary treatment of shock. FRIEDBERG, Chicago.

CLEFT PALATE AND THE MECHANISM OF SPEECH. MICHAEL C. OLDFIELD, Brit. J. Surg. **29**:197 (Oct.) 1941.

The author presents a well illustrated review of the anatomy of the palate and of the nasopharynx and emphasizes the importance of function in the repair of cleft palate. He recommends as the ideal time for operation between the ages of 1 and 2 years. The technic of the operation is discussed. He recommends treatment by one surgical unit, and the results in a conservative series of 120 cases are analyzed. The author points out that Strauss and Olson, who studied the congenital abnormalities of animals in the Berlin Zoo, made the observation that imperfect nutrition may be a causal factor in the production of cleft palate. They reported that in 1909, 32 jaguars, from the same dam and sire, were born with cleft palate. The diet was then changed and fresh meat given, after which no further offspring with cleft palate were born from the same parents. A somewhat similar experience was noted at Regent's Park Zoo, where cleft palate was common in lion cubs at the end of the last century. Since 1923, when the diet of lionesses was altered, only 2 litters have been born with cleft palates. The other important factor is the influence of heredity. In about 20 per cent of the cases a definite family history can be traced.

KEITH, Toronto, Canada [AM. J. DIS. CHILD.].

SULPHANILAMIDE AND SULPHAPYRIDINE IN EXPERIMENTAL CEREBRAL WOUNDS. E. H. BOTTERELL, E. A. CARMICHAEL and W. V. CONE, J. Neurol. & Psychiat. **4**:163 (July-Oct.) 1941.

When 500 Gm. of sulfanilamide and sulfapyridine crystals was placed in brain defects of 11 and 13 cats, respectively, the former drug disappeared in from eighteen to thirty-six days and the latter in from forty-four to fifty-eight days. In all cases the drug acted as a foreign body, causing a local inflammatory reaction, through which it was encapsulated and ultimately absorbed. In the early stages, the inflammatory reaction consisted of polymorphonuclear leukocytes and phagocytic cells, which invaded the crystals. Later, this was replaced by lymphocytes, plasma cells and giant cells. When absorption was complete, a scar of mixed neuroglia and connective tissue resulted. More polymorphonuclear leukocytes surrounded sulfanilamide in the early stages, and heavier encapsulation with connective tissue occurred about sulfapyridine. Because of its quicker absorption and the shorter period of the inflammatory reaction, sulfanilamide seems to be the drug of choice for local application in cerebral wounds. There were no changes in the nerve tissue at a distance from the drug, the reaction remaining purely local. The local scarring was no more extensive than that following wounds uncomplicated by the use of sulfanilamide or its derivative. The highest level of either drug found in the blood, resulting from absorption from the drug in the cerebral wounds, was not sufficient to be of therapeutic significance. There was no impairment in the general health of the animals and no pathologic changes in the internal organs which could be attributed to the drug. In no instance did postoperative infection occur, and the authors are of the opinion that use of sulfanilamide or a derivative is indicated in potentially infected or infected wounds, even though the bacteriostatic and bacteriolytic actions of such

drugs have not yet been conclusively proved. However, they advise caution, since the drugs act as foreign bodies and excessive quantities should never be used. Of practical significance is the effect of sulfapyridine crystals in controlling bleeding from smaller venous channels.

N. MALAMUD, Ann Arbor, Mich. [ARCH. NEUROL. & PSYCHIAT.]

THE TREATMENT OF BRAIN ABSCESS. D. W. C. NORTHFIELD, J. Neurol. & Psychiat. **5**:1 (Jan.-April) 1942.

Northfield reviews his treatment of abscess of the brain in 31 patients and finds that of 12 patients treated by drainage only 3 lived, whereas of 19 patients treated by aspiration, decompression or enucleation alone or combined 11 survived. Thus closed methods of treatment offer a much more favorable prognosis than the open method. Failures are primarily due to such complications as multiple abscesses, massive edema or acute meningitis. Edema may be controlled in the early stages by dehydration therapy, but as a rule the best results are achieved by surgical decompression. When meningitis, including ventriculitis, is a complication of brain abscess it should be treated by decompression measures rather than by frequent lumbar punctures, since the latter tend to promote herniation. Chemotherapy doubtless combats meningitis, but not the abscess proper. Successful treatment depends on accurate localization and a diagnostic burr hole, tapping or ventriculography. The time of surgical intervention varies but should be resorted to whenever the clinical condition begins to deteriorate. Aspiration should be attempted first. This is followed by decompression only if tension is not sufficiently relieved, if there is considerable swelling of the brain or if the abscess is deep seated. At the time of aspiration, the cavity of the abscess should be irrigated with a 50 per cent electrolytic solution of sodium hypochlorite. Repeated aspirations may be necessary, but intervals will gradually lengthen. Injection of 1 to 2 cc. of thorium dioxide into the abscess may be of help in roentgenologic visualization of the organization of the wall of the abscess. Enucleation of an abscess depends on the formation of a well defined and tough wall. This optimum period varies, but the usual time is about the third month. Enucleation may prevent the occurrence of a convulsive disorder, and anticonvulsants should always be administered until the abscess is fully eradicated. Drainage should be used only in exceptional cases, such as those of infected compound fractures of the skull or superficial subdural abscess. N. MALAMUD, Ann Arbor, Mich. [ARCH. NEUROL. & PSYCHIAT.]

DISCUSSION ON THE EFFECTS OF FLYING ON THE NOSE AND EAR. J. F. SIMPSON and others, Proc. Roy. Soc. Med. **35**:245 (Jan.) 1942.

Simpson stated that changes in altitude bring about the commonest disturbances of the ear and nose. Such changes may give pain in the region of the frontal sinuses, spreading toward the vertex, radiating between and behind the eyes and even involving the upper jaw. In case of the ear, in addition to pain, there are deafness and occasionally vertigo. The pain at times is intense and cannot be explained merely on the basis of passive congestion of the mucosa; other and more complex factors probably take part. An occasional complication of this aero-otitis is a severe and permanent form of nerve deafness without abnormal changes in the tympanic membrane or eustachian tube and without impairment of the vestibular function.

Besides alteration in altitude, the ear is affected by changes in speed, direction and position in aviation, particularly those due to vestibular reactions. Without adequate visual reference to the earth or instruments a pilot eventually and inevitably begins to fly in circles, has vertigo, goes into a spin and crashes. The development of the technic of blind flying has overcome this. Disease of the ear, nose and throat will impair the efficiency of an aviator, especially under conditions of modern aerial warfare, usually through deafness or predisposition to pain. All acute inflammatory conditions of the nose and throat can interfere with the mechanism for adjusting pressure in the ear and sinuses.

Dickson discussed the effects on the ear of noise experienced in aviation. The first evidence of such acoustic trauma manifests itself as a dip localized to 4096 cycles per second, both for air and for bone conduction. Repeated and continuous exposure to noise may involve frequencies below this level and give definite impairment of hearing. The factors which influence the degree of hearing loss from acoustic trauma are: (1) the total time of exposure; (2) the length of exposure at each period; (3) the character of the sound stimulus, whether continuous or interrupted; (4) the type of protection adopted; (5) the surroundings, whether in enclosed or in open spaces, and (6) previous disease of the ears. The wearing of a flying helmet with telephones attached affords the fullest protection.

Fry stated that hearing tests used at present for selection of flying personnel are not satisfactory and that he had experimented with a method of making a sound articulation test.

The subject is placed in a high level noise field, from which his ears are protected by the standard service flying helmet. Lists of words and sentences are then conveyed to him through a pair of telephone receivers carried in the helmet.

McGibbon discussed aviation pressure deafness, or aero-otitis. In a review of 100 cases the predominant symptom was deafness, which was often associated with pain, tinnitus or vertigo. Normal drum heads were present in 26 cases; invagination of the tympanic membrane was noted in 47 cases; congestion of the attic, in 6 cases; congestion of the entire drum head, in 8 cases; effusion into the tympanum, in 6 cases, and rupture of the drum head, in 7 cases.

In discussion of inflation of the eustachian tube, consideration was given to the pressure that could be created in the nasopharyngeal and nasal cavities by various means, such as Valsalva's method, the Politzer bag and the eustachian catheter. The anatomy of the parts involved and the physiology of the eustachian tube were also reviewed, and the action of the tube during quick ascent and descent in an aircraft was described, as well as the effect on the ear when there is failure of the tube to open properly.

Winfield discussed the causation of air sickness from the physiologic, pathologic and psychologic points of view. The treatment of air sickness is directed mainly toward taking care of such accessory factors as ventilation, comfort, diet, noise and vibration and ocular stimuli.

CAMPBELL, Philadelphia.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

G. HENRY MUNDT, M.D., *President*

Regular Meeting, Jan. 4, 1943

Primary Carcinoma of the Eustachian Tube: A Study of Evidence of Its Occurrence. DR. LAWRENCE J. LAWSON, Evanston, Ill.

This tumor is to be distinguished from secondary involvements of the eustachian tube by a neoplasm originating in some other nasopharyngeal location. It is the purpose of this paper to review the clinical features and the literature and to report a new case of such a tumor discovered during life, with comments on therapy.

The symptoms fall essentially into three stages: (1) obstruction of the eustachian tube, with tinnitus and deafness; (2) development of a nasopharyngeal tumor, and (3) intracranial invasion through the basal foramens.

The literature contains several well authenticated cases, including a few in which the diagnosis was sarcoma, some of which, with the present understanding of the pathology of this tumor, might possibly be reclassified as transitional cell carcinoma.

REPORT OF A CASE

A man aged 59 was treated after symptoms of the second stage were present. He gave a clear history of obstruction of the eustachian tube for many months before the development of symptoms of tumor. The cervical glands were not involved. High voltage roentgen therapy was combined with implantation of radon. Biopsy revealed transitional cell carcinoma. Nine months later the patient is in good health and free from symptoms of tumor. Sufficient time has not elapsed for this patient to be reported as cured.

There must be close cooperation between the radiologist and the otolaryngologist, so that the patient will not be deprived of therapeutic opportunities. The most favorable treatment appears to be combined high voltage roentgen irradiation and implantation of radon. The early symptoms are not disabling; therefore when the patient is seen late, effective control of the neoplasm is much less likely. Patients with more recent growths treated in accordance with the Coutard principles of irradiation appear to survive longer.

DISCUSSION

DR. JOHN F. DELPH: In studying my own cases and those of the clinic, I have been struck by the lack of early symptoms. In practically all cases the tumor was well advanced and multiple cervical metastases, hemorrhages, anemia and, in some cases, neurologic signs of cranial invasion were present. From the history one may often learn of symptoms which may be construed as early signs of a malignant growth in the epipharynx, although not in themselves indicative of such a tumor. Disturbances of the olfactory organ were noted in the complaint of fetor or complete anosmia. In practically all cases there was mention of a nasal or postnasal discharge and nasal blockage of some degree. Repeated small spontaneous nasal hemorrhages, recurring intermittently over a period of months, or one or two massive losses of blood requiring hospitalization were noted. Fulness of the ear and tinnitus, mentioned by Dr. Lawson, were practically absent in the series of cases studied. Disturbance along the distribution of the trigeminal nerve, giving rise to mild pain, was sometimes complained of. Martin described this pain as usually deep-seated, poorly localized distress, which is persistent, but occasionally paroxysmal. Other symptoms were a feeling of thickness in the soft palate, a nasal quality to the speech and disturbance of deglutition. In 1 case the first sign noted was a seropurulent discharge from the middle ear. Because of the tendency of this tumor to spread by contiguity and to form early metastases, the floor of the cranial cavity is frequently invaded, and the early signs then become neurologic. The third, fourth and sixth nerves are usually the first to be affected, with resultant diplopia; complete

ptosis and ophthalmoplegia are later signs. Gradenigo's syndrome was mentioned; this, with a discharging ear, may cause confusion in diagnosis. Tinnitus and vertigo are often early indications of cranial invasion. Nausea and vomiting with deglutition speak for extension along the brain stem.

The aforementioned symptoms and signs have been gathered from a series of cases of proved lymphoepithelioma of the nasopharynx. They are not restricted to malignant growths; a number of them are common to less serious lesions of the nose and throat. True, the prodromal symptoms are few and meager, but the occasional spitting of a small quantity of blood, nasal blockage or persistent nasal discharge calls for more thorough study of the nasopharynx than is generally given by the busy otolaryngologist. This can best be made through direct visualization by means of a palate retractor or the nasopharyngoscope.

I should like to report the case of a man seen in consultation with the neurosurgical service, in which one of the early symptoms was referable to the ear.

R. C., a white man aged 39, was first seen because of diminution of hearing and a sensation that the left ear was plugged for five weeks, pain about the right eye for five weeks, numbness of the skin over the right eye for two weeks, diplopia on looking to the right for three weeks and difficulty in swallowing. A nasal discharge of mucopus had been present for five weeks. Neurologic examination showed widening of the left palpebral fissure, complete paralysis of the right lateral rectus muscle, nystagmus on looking to the left, anesthesia of the right maxillary area, diminution of the right corneal reflex, deafness in the left ear, a voice of nasal quality and paralysis of the left side of the soft palate. There was no complaint of headache. Examination of the ears showed a sclerotic drum membrane on both sides, with an old perforation in the right one. In the vault of the nasopharynx, obstructing the orifice of the left eustachian tube, was a mulberry-like mass of nodular tissue, the size of a large olive, and extending from it across the roof was an apron of the same tissue. Large vessels were discernible. Report of biopsy stated that there was dense lymphoid infiltration, with chronic inflammation. Because of the carcinoma-like appearance and the undoubted intracranial extension, the patient was immediately given a course of radium treatments. A total exposure of 300,000 milligram hours was given, the radium being used in the form of external packs. Three and a half months after the beginning of treatment only a small nubbin of the tumor was left, and most of the neurologic symptoms had disappeared. The patient is now in perfect health, three years after treatment, with no vestige of intracranial extension, and the mucosa in the epipharynx is smooth and clean.

This man undoubtedly had a visible nasopharyngeal tumor before intracranial extension developed, for the complaints were indicative, i. e., diminution of hearing and a feeling that the ear was blocked. The case illustrates that the most prominent symptom, paralysis of the right lateral rectus muscle, may be contralateral to the primary tumor and, also, that the symptoms may be of short duration. Davis and Martin have previously reported this case (*Ann. Surg.* **112**:1058 [Dec.] 1940).

Dr. George H. Woodruff, Joliet, Ill.: I wish to mention 2 cases of carcinoma which originated about the orifice of the eustachian tube. The first was that of a youth aged 20 years. The tumor apparently originated in the fossa of Rosenmüller and was about the size of an olive pit. Symptoms were referred to the right ear, with blockage of the eustachian tube, tinnitus, loss of hearing and pain over the side of the face. No involvement of the cervical glands was evident. The boy had had treatment elsewhere, in an effort to get relief from the obstruction. The tumor was discovered with the nasopharyngoscope. The patient was referred to Dr. Cutler about seven years ago for treatment with radium. He is alive and well today, without signs of recurrence; there is dryness of the nasopharynx.

In the second case the carcinoma was more advanced. The orifice of the eustachian tube was covered by the tumor. Where it originated I cannot say. There were palpable glands in the neck. As in the first case, the diagnosis was transitional cell carcinoma. This patient also was treated with radium by Dr. Cutler and is well today, about five years after he was first seen.

Dr. Arthur J. Coombs: While I have not seen many malignant tumors in this region, I shall mention 2 cases. In the first case, a man aged 46 had previously been operated on because of bleeding from the tonsil. When he came to me I discovered a large nasopharyngeal tumor in Rosenmüller's fossa. Biopsy revealed transitional cell carcinoma. There were involvement of the cervical glands on the same side, slight diminution of hearing and tinnitus and, later, otitis media and pain. He was treated by the fractional method of roentgen irradiation, and apparently the tumor disappeared. About nine months later it recurred. Further treatment was of no avail, although given over the glands of the body. He died of generalized metastases about two years after the tumor was first discovered.

In the other case, a woman aged about 32 is now under care at the Illinois Research and Educational Hospital. When she was first seen, last spring, a definite mass, measuring about 4 by 8 or 10 mm., protruded into the nasopharynx. With roentgen therapy the tumor disappeared rapidly. There was recurrence this fall, and she is now receiving further roentgen irradiation. The radiologist advised against the use of radium. Convergent strabismus developed, involving the side of the tumor, and a roentgenogram showed that the lesion invaded the petrous portion of the temporal bone. In both these cases there was remarkable resolution of the process with roentgen ray therapy, but subsequently recurrence developed at the same site in both cases.

DR. GLENN J. GREENWOOD: How effectively was the pain controlled by radium therapy? In the old days, alcohol was injected into the second or third division of the fifth nerve to control the distressing pain from a nasopharyngeal new growth.

DR. LAWRENCE J. LAWSON, Evanston, Ill.: The purpose of this paper was to emphasize two points: First, the otolaryngologist should make early careful and repeated examinations for this inaccessible tumor; second, the patient should be treated early and persistently by an experienced radiologist, working in close cooperation with the otolaryngologist. If this cooperation is lacking, therapeutic opportunities may be lost. Better types of radiation therapy are now available.

The patient on whom I reported has not had the severe pain that is produced later in the invasive stage.

Neoplasms of the Ear, Nose and Throat: Clinical Report of Cases. DR. L. B. BERNHEIMER.

Clinical reports of several neoplasms of the ear, nose and throat were presented, with lantern slides. These neoplasms include carcinoma of the lip, adamantinoma of the mandible, carcinoma of the floor of the mouth, carcinoma of the tongue, tobacco chewer's carcinoma of the buccal surface of the cheek, carcinoma of the tonsil, carcinoma of the larynx, metastatic melanoma of the larynx, chondroma of the larynx and carcinoma of the middle ear.

Methods of treatment were discussed, and clinical problems, such as prophylactic dissection of the neck in cases of carcinoma of the lip, the choice of irradiation rather than of operation for noninfiltrating lesions of the larynx and the end results in treatment of the types of tumors demonstrated, were emphasized.

DISCUSSION

DR. SAMUEL SALINGER: Dr. Bernheimer has presented an interesting array of cases. In the main, I can agree with everything he has said about therapy. Some of these tumors are rare—adamantinoma, for example. I believe I have seen 2 patients with this neoplasm, the tumor involving the superior maxilla in both instances. One of these patients is well, after three years, and the other has been lost track of.

In my experience, melanoma is one of the most tragic conditions with which one has to deal. I have never known of a patient's being cured, and the head of the tumor clinic with which I am associated, who has seen many cases of the tumor, knows of only 1 cure. Since the tumor is radioresistant, the only chance for cure is radical operation. I am keeping under observation a young woman with a small lesion on the cheek which for two years has been stationary. When I first saw it, I advised radical resection, with removal of part of the cheek and nose, but the patient objected. No one can predict the outcome, since it is well known that the lesion may remain quiescent for a long time and suddenly become active.

Dr. Bernheimer's experience with carcinoma of the tonsil is contrary to that of other observers. Most authorities who have access to a large amount of statistical data feel that carcinoma of the tonsil is one of the most amenable to therapy of all malignant growths about the head because of the lymphoid nature of the tumor and its bed. I agree, however, that the presence of metastases diminishes the chance of cure. In the few cases I have seen the growth yielded promptly to irradiation and has shown no recurrence for years.

DR. THOMAS C. GALLOWAY, Evanston, Ill.: All agree with the methods of treatment outlined by Dr. Bernheimer. Even ten years ago there was much disagreement as to treatment in any given case; now the therapy seems rather well standardized. In the old days I employed coagulation for many cancers of the tonsil, with probably only 1 cure. Statistics show that about 24 to 28 per cent of such neoplasms are curable with radiotherapy, which is the method of choice.

Adamantinoma, or ameloblastoma, occurs more frequently in the mandible, but in the maxilla it is harder to control. It is a deceptive tumor; even though relatively benign, it

creeps along the periosteum until it gets into the orbit or the cranium, and then the outlook is bad. I have a nine year cure in 1 case; in 3 others the outcome was fatal. As to the glands in cases of cancer of the tongue or cheek, at a symposium of the American Radiological Association nearly every one present agreed that block dissection was indicated early, or when the first sign of a malignant growth developed. When the glands are more than 1 cm. in diameter, the prognosis in most instances is grave.

The malignant nature of melanoma is well known; yet I saw a patient twenty-one years ago whose tumor was treated elsewhere as a cavernous angioma, but was subsequently found to be a melanosarcoma. I saw him recently; he had multiple recurrences all over the forehead and in the nose, and yet he was carrying on his business.

Bronchogenic Carcinoma: Analysis of One Hundred and Twenty-Five Consecutive Cases. DR. PAUL H. HOLINGER and DR. H. JAMES HARA, Hinsdale, Ill.

Analysis of 125 consecutive cases of bronchogenic carcinoma studied at the Research and Educational Hospital and at St. Luke's Hospital shows an increasing incidence with each succeeding year. The series included 107 males and 18 females; 123 of the patients were white persons; 1 was a Negro, and 1, a Filipino. The oldest patient was 76 and the youngest 6 years of age, with the greatest number in the age group of 55 to 59 years.

The significant symptoms were cough, hemoptysis, loss of weight and, in almost every case, pain or "discomfort" in the chest. Pneumonia, which was "unresolved," was common. Hoarseness was the first symptom noted in 7 cases; in 5 cases the left, and in 2 cases the right, uveal cord, was paralyzed.

In 98, or 78.4 per cent, of the cases biopsy of a specimen obtained with the bronchoscope revealed the presence of carcinoma, and in 7 additional cases evidence was obtained by exploratory thoracotomy, examination of sputum or aspiration of fluid from the chest. In the majority of cases bronchoscopic examination showed an intraluminal tumor, an irregular, freely bleeding bronchial wall or definite infiltration of the mucosa. However, in patients from whom it was impossible to obtain tissue for biopsy, confirmatory information was obtained.

Inability to obtain tissue from a representative area is due to variable factors. The tumor may be too far in the periphery or in the upper lobe of the lung, or a tumor with extension into the bronchus peripherally may compress the bronchus proximal to that invasion and prevent passage of forceps to the intraluminal lesion. Not infrequently, too, a carcinoma so invades the mediastinum that satisfactory examination of the bronchi is impossible, owing to the "frozen" character of the tracheobronchial tree.

Squamous cell carcinoma was the most common. Adenocarcinoma was least common, and undifferentiated carcinomas were between the two extremes in frequency. The sites of the lesions were as follows:

Location	No. of Cases
Right main bronchus	45
Bronchus of upper lobe of right lung	10
Bronchus of lower lobe of right lung	29
Periphery (not visualized)	3
Total on right side	87
Main bronchus of left lung	12
Bronchus of upper lobe of left lung	8
Bronchus of lower lobe of left lung	15
Periphery (not visualized)	2
Total on left side	37
Bilateral	1
Total	125

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLARYNGOLOGY, AND PHILADELPHIA LARYNGOLOGICAL SOCIETY

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Regular Meeting, Jan. 20, 1943

GEORGE L. WHELAN, M.D., *Editor of Abstracts*

Analysis of One Hundred Consecutive Cases of Aural Disease in an Army General Hospital. CAPTAIN JOHN J. CONLEY, Medical Corps, Army of the United States (by invitation).

This article will be published in full in a later issue of the ARCHIVES.

DISCUSSION

DR. GEORGE M. COATES: It is encouraging to know of the good care the men are getting in the Army hospitals. It is also comforting to know that competent medical men are looking after them when they get into trouble. Army hospitals are far better and more fully equipped today than they were during World War I. They are even better than are civilian hospitals.

How do patients suffering from chronic purulent otitis media get into the Army in the first place? They must have had the disease before induction. It may have been the fault of the examiner.

During World War I there were not the present facilities for taking care of aural disorders. Radical surgical procedures were not permitted. Local anesthesia was not employed. If the patient did not respond to therapy he was sent back to civilian life.

Many soldiers have acute mastoiditis due to running ears and infections of the respiratory tract. Under present conditions there is a much better chance of curing these disorders.

From Captain Conley's review, it is apparent that all patients with nonsurgical conditions had prolonged treatment with sulfonamide compounds. Captain Conley prefers the use of sulfanilamide. My experience with sulfathiazole has been satisfactory. Fenton prefers sulfathiazole, too.

I am glad to know that Dr. Conley is using the Lempert technic. It is the young man's method of doing a mastoidectomy. I believe it is the approach of the future.

DR. HORACE J. WILLIAMS: Captain Conley's analysis of 100 cases of aural disease is excellent. That 4.3 per cent of the first 2,290 patients admitted to the hospital should have some form of aural involvement is more than one would expect. In 59 per cent of the 100 cases the complaint was chronic suppurative otitis media.

During the last fifteen years the otologist, the pediatrician and the general practitioner have had a much better understanding of the acute aural infections, and, as a result, there has been a gradual decrease in the incidence of chronic suppurative otitis media and mastoiditis in the urban cities. A large percentage of patients with chronic suppurative otitis operated on in one of the hospitals with which I am connected come from the rural districts, where free clinics are not available.

Local therapy with the sulfonamide compounds represents the best conservative treatment of chronic suppurative otitis media. My experience with the local use of such drugs has been more than satisfactory, and sometimes surprising. Two patients treated with sulfanilamide had a reaction in which there was a profuse watery discharge. A change to sulfathiazole decreased the discharge, but the ears did not become dry.

Suppurative otitis media, with a central perforation of the pars tensa of the tympanic membrane, varying in size, form and location, reacts favorably to local application of a sulfonamide compound. Reinfection of the nasopharynx will probably lead to reinfection of the middle ear. This is the nondangerous type of chronic suppurative otitis media.

When the perforation is in Shrapnell's membrane or in the margin of the membrana tensa, a sulfonamide compound usually produces a dry ear in a reasonable length of time. Cholesteatoma in the attic and the mastoid antrum can hardly be expected to respond permanently to treatment with a sulfonamide compound unless the drug is introduced through the perforation and is diffused through the attic and the mastoid antrum.

The length of time that the tympanic cavity remains dry will be determined later, when many reports from various sections of the country are reviewed.

Grafts as a means of decreasing the healing time of radical mastoidectomy cavities are worthy of use. About one year ago I began using skin grafts in such cavities. In the first 2 cases grafts were placed only in the tympanic cavity. These grafts lived, and the tympanic cavity dermatized quickly. In later operations several grafts were placed in various parts of the tympanomastoid cavity.

Method of Use of Grafts.—The radical mastoidectomy cavity is thoroughly cleansed with a warm solution of sodium chloride, after complete removal of all infected bone and tissue. The tympanic cavity is then packed with gauze soaked in a solution of epinephrine to control all bleeding, so that the graft may be placed on the bony surface free from blood. The presence of any foreign body, such as blood between the graft and the surface on which it lies, prevents the adherence of the graft to the bone, and it will die. When the graft is first placed on the bone, it exists only by the absorption of lymph from the underlying surface, which the presence of a foreign body would prevent.

A split graft was used in all but 1 case. The graft is cut with a long, thin, sharp knife. The skin is held taut by pressure on small sterile blocks of wood placed on each side of the area to be cut. The knife is made to penetrate to the desired depth, and with a to and fro motion the graft is cut easily. The area on which the graft is to be placed has previously been made ready to receive the graft. A piece is cut from the large graft the size to cover the area on which it is to be placed. The graft being thin, it rolls when picked up and resembles a string. To prevent rolling, the four corners of the cut graft are seized with forceps, and the graft is doubled, with the skin surfaces together, which makes it easy to place in the tympanomastoid cavity. Small pieces of 0.25 inch (0.64 cm.) selvage edge gauze are used to make pressure on the graft so that it will fit into all corners. The first two or three pieces of packing are tied with black silk, so that the packing will be easily identified. The graft must be held in place and accurately approximated to the surface in which it is placed by an even pressure to insure the adherence and life of the graft. The packing is permitted to remain in the wound ten to twelve days unless a foul-smelling discharge develops. The outer pieces may be removed, and when the packing near the graft is reached one of the sulfonamide compounds may be placed on it. This aids in keeping the wound free from infection. After all the packing has been removed, it is repacked only when areas of granulation make their appearance between the grafts.

Two cases are of particular interest. F. R., a man aged 35, had had chronic suppurative otitis media since childhood. A radical mastoidectomy was performed on the left side. A thin graft was placed in the tympanic cavity and over the facial ridge. On the tenth day signs of pulmonary abscess developed. This complication was confirmed roentgenographically. In less than three weeks the entire tympanomastoid cavity had dermatized completely. His ear was not examined for about ten days after it had healed. During this time an acute cold developed, followed by an acute flare-up of an old chronic maxillary sinusitis on the side of the mastoidectomy. Some infection gained access to the eustachian tube, and, through the patient's coughing and blowing his nose, this infection was forced into the tympanomastoid cavity. Some granulations and discharge developed in the region of the opening of the eustachian tube. Sulfanilamide was placed in the wound and the opening closed with packing, and the cavity has remained dry.

B. H., a Negress aged 25, had had a discharging ear since childhood. The Wassermann reaction was 4 plus. A radical mastoidectomy was performed.

The entire tympanomastoid cavity was covered with thin grafts. On the third day after the operation the discharge from the wound was so profuse and the odor so offensive that the packing was removed. The profuse, foul discharge persisted; the odor was objectionable, and part of the postauricular incision opened. Through the external auditory canal, the wound was irrigated with a solution of potassium permanganate and then with sterile water and dried. Sulfanilamide powder was placed in the cavity. The discharge ceased in about a week. All the grafts appeared to be holding. In the tympanic cavity part of the graft seemed to be separating from the superior wall. This was watched daily and finally was taken hold of with a pair of forceps. It proved to be a piece of packing. Beneath this packing the graft had disappeared. In a few days the area was dermatized from the adjacent grafts. Only once was a secondary graft used. It proved also to be satisfactory.

Orbital, Osseous and Intracranial Complications of Nasal Origin. LIEUTENANT COMMANDER FRANCIS LEDERER (M.C.), U.S.N.R. (by invitation).

The protein manifestations, etiologic factors, routes of extension and combined clinical pictures of naso-orbital infections arouse the interest of the ophthalmologist, the otolaryngologist, the neurologist and even the dental surgeon. The subject is much broader than a cursory consideration of the title indicates. Included in the problem is the necessity of a thorough appreciation of anatomic configurations leading to infection through venous and lymphatic channels, along fascial planes, by continuity and contiguity of structure, by foramens and dehiscences and by perineural sheaths. Such routes of extension may bring about four main clinical groups: (1) naso-orbital; (2) nasocerebral; (3) orbitocerebral, and (4) naso-orbitocerebral.

Osteomyelitis.—This process cannot be considered adequately without defining its anatomic site, the type of pathologic process, the duration of the disease (whether acute, chronic or latent) and the degree of infection of the brain. Osteomyelitis of the sphenoid sinus merits special mention because it may produce a confusing clinical picture. It is usually accompanied by headache, either frontal or occipital or both, by slight rigidity of the neck and by occasional vomiting. Such symptoms are also observed in occasional cases of uncomplicated sphenoid sinusitis or basilar meningitis. The persistence of mild meningitic signs, in addition to evidence of sinusitis and the signs of infection in the spinal fluid, point to involvement of the sphenoid sinus. The ears are frequently a cause of confusion, and occasionally otologic symptoms prevail to such a degree that the mastoid becomes the site of surgical intervention.

Treatment: The management of osteomyelitis of the skull is both medical and surgical, but there is no stereotyped therapy. The time for surgical intervention in cases of osteomyelitis of the skull is uncertain, and the experience in one case does not always aid in the conduct of the succeeding one. Early recognition is stressed but is clinically difficult. Treatment must be individualized. A formula suggesting the optimum time for operation, unfortunately, is lacking. Essentially, before an area of localization is determined, application of infra-red heat, short wave therapy and roentgen radiation to the affected area may hasten the process. While in some instances simple drainage of a localized abscess and expectant treatment with regard to sequestrums have sufficed, a more adequate attack is frequently essential. This consists in thorough and wide removal of all diseased and a portion of the surrounding healthy bone. All necrotic tissue must be removed and the cavity left wide open for drainage. Cosmesis should be completely disregarded. In cases in which the frontal sinus is involved, exposure of the frontal lobe of the brain frequently becomes necessary. Covering skin flaps should not immediately be replaced over the dural areas because of the possibility of locking up a space which would enclose an abscess. Supportive agents include blood transfusions, tonics, nutritious food and an improved hygienic regimen. Chemotherapy, given locally as well as parenterally, has not proved effective and may mask or confound the interpretation of otherwise helpful signs and symptoms.

The question arises as to when osteomyelitis is to be considered cured. Latency, so characteristic of the disease, encephalitis, abscess of the brain, metastases to other osseous structures of the body, parenchymatous degeneration of remote organs and jacksonian epilepsy following removal of portions of the skull, all influence the ultimate prognosis. It is to be emphasized that in one third of the cases in a recent series metastases developed in a year and a half to thirteen years after the onset of the original process. Because in all these cases evidence of defects close to the midline neighboring the longitudinal sinus existed, it is possible that the multiple metastases were the result of a thrombotic process.

Rhinogenic Abscess of the Brain.—From the aforementioned etiologic factors and pathways, it is readily understood how infection of the frontal, the sphenoid and occasionally the ethmoid and maxillary sinuses may lead to the formation of an abscess of the brain. Abscess formation is usually associated with a purulent process traveling by direct osseous extension. The necrotic process of the frontal sinus produces an abscess of the frontal lobe, and that of the sphenoid sinus, a basilar abscess of the frontal, or occasionally the temporal, lobe. Extension from the orbit and the roof of the nose and spread by way of venous channels have already been alluded to.

In the early stages of a brain abscess symptoms may be only obscure, or not exist at all. Their presence is difficult to determine at times. Suffice it that manifestations are dependent on the causative factor, the age of the patient, the severity of the infection and the site and the stage of the abscess with reference to its duration. These factors determine to a great extent the symptoms which may be associated with the lesion. Drowsiness with stupor, delirium, fever, chills, nuchal rigidity, muscular twitchings and convulsions are commonly encountered with acute abscess. Central anosmia seldom occurs. Headache, vomiting and signs of rapidly increasing intracranial pressure are usually present. Perhaps the most reliable neurologic

sign of a lesion of the frontal lobe is weakness of the opposite side of the face of supranuclear type. The abscess is usually large, and papilledema of some degree is frequently present. Ventriculograms and encephalograms may yield information of considerable value.

Treatment: All the aforementioned factors influence the treatment of rhinogenic abscess of the brain. Before the treatment of the abscess itself is considered, the management of the primary focus is frequently necessary. It is best not to interfere in the acute stage of an abscess, but to eradicate the primary focus by facilitating drainage, treating the sepsis by means of chemotherapy, giving frequent small blood transfusions and awaiting localizing signs or encapsulation. Technics for the drainage of an abscess vary with the surgeon, but questions of the optimum time for intervention and whether or not one should approach the abscess by a clean field are still moot. Given a patient who has been fortunate enough to survive the invasion of the meninges or encephalitis, the field may be approached through a so-called uninfected area, with the future possibility that the primary focus may be cleared up later. This may not prove advantageous, however, for the focus will continue to infect the brain. This is especially true when there is associated osteomyelitis. Furthermore, to enter through an area other than the primary focus may precipitate further osteomyelitic extension. It may be emphasized here that there are few proved cases in which infection was actually carried to the brain by operation through an infected field. If the surgeon can await encapsulation, the abscess may be managed by a number of recognized methods, viz., the open or the closed method, extirpation in toto or tapping. In the great majority of cases, the abscess will be found in the immediate neighborhood of the primary focus; rarely does it occur as a heterolateral process or in some other part of the brain.

DISCUSSION

DR. OSCAR V. BATSON: By slides made in the anatomic laboratory, opaque fluid injected into surface veins was ultimately traced into the intracranial veins and sinuses.

DR. OTTO C. HIRST: Dr. Lederer has given his usual splendid, comprehensive presentation, leaving little to be added or discussed. I may, however, emphasize certain salient points and state a few aphorisms based on personal experience.

In the light of present knowledge, there is little difference of opinion as to the wisdom of radical surgical intervention in osseous complications of sinus origin. In 1938 there was a wide divergence of opinion. I still believe, as I stated previously (Osteomyelitis of the Skull Complicating Mastoiditis and Frontal Sinusitis, ARCH. OTOLARYNG. **29**:24 [Jan.] 1939), that the current of infection is more inward than outward, that it is more important to recognize the possibilities of diffusion than to spend time and effort in a differentiation of types of osteomyelitis. Once a diagnosis has been established, radical operation, with wide resection of bone, should be carried out. This removal of a focus of infection, with invasion of the intracranial contents in mind, is based on good surgical principles, for one may expect any osteomyelitic lesion in the skull to cover an area of pachymeningitis or an extradural abscess. Furthermore, if the condition is complicated by a subdural abscess or an abscess of the frontal lobe, the decompression will permit better localization of the abscess and, likewise, prevent compression of the brain and its vital centers.

As to the orbital complications, the two commonly seen, cellulitis or abscess formation and acute retrobulbar neuritis, are interesting opposites. In my experience, cellulitis or abscess formation is usually seen in children, after infection of the anterior series of sinuses, and usually responds to conservative treatment or simple external incision. Radical surgical intervention is usually contraindicated. I have seen meningitis in an infant follow infraction of the middle turbinate for acute purulent ethmoiditis, complicated by orbital cellulitis.

Acute retrobulbar neuritis, on the other hand, is usually seen in adults, after infection of the posterior series of sinuses, and demands immediate radical treatment. From my experience, I do not believe that all acute retrobulbar neuritis is a sign of multiple sclerosis. I operate first and then ask for a neurologic study.

My experience with intracranial complications of sinus origin has been limited to those following acute fulminating frontal sinusitis and to a case of protective meningitis complicating acute purulent sphenoiditis. The sphenoiditis was part of acute pansinusitis complicating a submucous resection. The patient recovered, and it was interesting to note the improvement in the meningeal symptoms with each daily lavage of the sphenoid sinus.

The complications under discussion occur despite good surgical treatment and extensive experience. However, a few aphorisms may be appropriate at this time. I base them on two lines of thought. First, in general, an acute infectious nasal lesion is more apt to result in an intracranial complication than is a chronic one, a situation which differs from that present in otitis media or mastoiditis, in which chronic disease is more likely to extend intracranially. Second, in chronic conditions of the sinuses, just as in chronic conditions of the ear, the inflam-

mation is not limited to the mucosa. There is an underlying pathologic osteitis or osteomyelitis, due to proximity of structure. This pathologic osteitis or osteomyelitis awaits favorable conditions to become a clinical condition.

In our clinic my associates and I observe the following rules for the prevention of such tragic sequelae of sinus disease as osteomyelitis and meningitis: 1. Rasping operations on the frontal sinus are not permitted. They grind infection into the bone.

2. Sharp, clean cutting and biting instruments should be used.

3. Sulfonamide compounds in proper dosage are given in preparation for all operations on the sinuses and for minor operations on the nose in the presence of acute sinus infection.

4. Whenever possible, bone work in the treatment of acute sinusitis should be avoided. If, however, it is deemed necessary to intervene surgically in a case of acute purulent frontal sinusitis, the operation should be an external frontal sinuotomy, rather than a middle turbinectomy. We believe that the external approach is less hazardous than that in the region of the cribriform plate or the fovea ethmoidalis.

5. The external approach to the acutely infected frontal sinus should be through the inferior, and not the anterior, wall, as was formerly taught and is sometimes still practiced. This is because the inferior wall is made up of compact bone, and the lower portion of the anterior wall is composed of diploic bone, which is the ideal type for the starting place of osteomyelitis. Furthermore, drainage is more direct through the inferior wall.

6. Patients with meningitis, osteomyelitis or acute purulent sinus conditions needing operation are all dehydrated and have high blood sugars. Therefore we prepare them before operation either with blood transfusions or by administration of a 5 per cent solution of dextrose, to which 15 units of insulin is added.

7. We do not perform a radical operation during an acute sinus infection or during an acute exacerbation of a chronic condition. I am convinced that it is no mere coincidence that 72 per cent of cases of cranial osteomyelitis follow an operation performed during an acute phase of sinus infection.

DR. BENJAMIN H. SHUSTER: The pictures shown by Dr. Lederer make one fearful about practicing otolaryngology at all. It seems that extensive osteomyelitis and orbital involvement may follow even some slight procedure. Fortunately, this does not happen often.

In spite of the various pathways of infection which lead intracranially, one still lives a physiologic life. By that I mean that one localizes and battles infection, and it is only in rare instances that the infection assumes ascendancy over the resistance, with the results illustrated.

Treatment of these extensive lesions varies from a mild surgical procedure to the most extensive removal of cranial bone. However, the surgical method, which at present is the best available, will not necessarily be the solution to the problem. Apparently, a certain strain of organisms is responsible for these complications. If only some laboratory will develop a chemotherapeutic agent to combat these organisms, much of the extensive surgical intervention probably will not be necessary. Simple drainage will suffice.

I wish to express my appreciation of Dr. Lederer's treatment of this subject. He approached it not from the point of view of a particular lesion but from the generalized standpoint of the many things that can happen in conjunction with sinusitis—and all things must be thought of. In the individual case, disease does not follow the textbook description.

Book Reviews

The Inner Ear. By Joseph Fischer, M.D. and Louis E. Wolfson, M.D. Price $5.75. Pp. 430, with 79 illustrations, 4 in color. New York: Grune & Stratton, Inc., 1943.

This book, as pointed out by the authors in the preface, is an attempt to bridge the gap between the usual textbook treatment of this subject and the voluminous material found in encyclopedias. The volume is concerned chiefly with the labyrinth and its central pathways and its relation to otoneurology, otosurgery and problems in modern warfare. As the authors point out, almost every topic is considered twice—once in the chapters on physiology, in which the various theories and hypotheses are discussed and references to the bibliographic material are noted, and again in the chapter on functional tests, in which indications for and the technic, clinical significance and evaluation of the various tests are critically reviewed. In this manner, each reader can find just what he is looking for, without having to read through pages of theory and literature.

The first two chapters are devoted to a brief review of the clinical anatomy and general physiology of the labyrinth. Chapter 3 is concerned with applied physiology and discusses the spontaneous and induced reactions of the labyrinth. Chapter 4 is an excellent résumé of the functional vestibular tests, with a discussion of spontaneous and induced manifestations and an evaluation and interpretation of abnormal reactions. Chapter 5 deals with the primary diseases of the labyrinthine capsule and is an excellent presentation of otosclerosis. Its exposition of the pathologic anatomy of this disease is noteworthy, and the material is illustrated with excellent photomicrographs. The various etiologic theories are well covered in a few words. Chapter 6 is concerned with inflammatory diseases of the labyrinth and of the petrous pyramid. A new classification of labyrinthitis is presented, based on that used by Alexander in Vienna. Indications for surgical treatment and operative technic are discussed. In chapter 7 the intracranial complications of labyrinthine infection and abscess of the cerebellum and temporosphenoidal lobe, meningitis, sinus thrombosis and subdural and extradural abscess are considered. Chapter 8 deals entirely with chemotherapy. Chapter 9, on facial palsy, is concerned with the anatomic and physiologic basis, classification, etiologic factors, clinical signs, symptoms and treatment. Chapter 10, on congenital diseases, is concerned with changes in the inner ear and in the neural and sensory apparatus. Chapter 11, on neoplasms, is concerned with tumors of the inner ear and of the cerebellopontile angle and with tumors of the anterior and middle cranial fossae. Chapter 12, on vascular lesions, is an excellent résumé of Ménière's syndrome. Chapter 13, on war trauma, is concerned with fractures of the skull, concussion of the inner ear, trauma due to explosion and gunshot injuries. Chapter 14, on the role of the inner ear in aeronautics, deals with the physiology of the cochlea and labyrinth in normal environment, as well as under unusual conditions, such as those of increased or decreased atmospheric pressure. Chapter 15 is concerned with the effects of atmospheric pressure on the ear.

All practitioners and graduate students in otolaryngology will find this book valuable. The authors are to be congratulated on their simple and concise presentation of a difficult subject. Each chapter is well organized and is easy to read. The bibliographic references at the end of each chapter are comprehensive and well chosen. The last three chapters, those on war trauma, the role of the inner ear in aeronautics and the effects of changes in atmospheric pressure on the ear, are timely.

One might question the wisdom of incorporating a chapter on chemotherapy in a book of this kind. This subject is in such flux at present that by the time the book is published the material is outdated. The chapter dealing with this topic is the only weak point in the book. For example, the authors state: "Sulfadiazine is poorly soluble and is used mostly as a local disinfectant in dysentery and similar conditions, and is not of great importance for further discussion here." However, in fairness to the authors, they state later in the chapter that at present the chemotherapeutic agent of choice for the majority of infections is sulfadiazine. This chapter, however, does not detract from the value of the book, although it were better omitted. A chapter on chemotherapy can scarcely be considered appropriate in a treatise on the labyrinth.

Directory of Otolaryngologic Societies *

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American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Claude C. Cody, 1304 Walker Ave., Houston, Texas.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.

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President: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
President-Elect: Dr. Lawrence T. Post, 508 N. Grand Blvd. St. Louis, Mo.
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Place: Chicago. Time: Oct. 10-13, 1943.

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President: Dr. Robert L. Moorhead, 125 Ramsen St., Brooklyn.
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American Laryngological Association

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Secretary: Dr. Isidore Friesner, 101 E. 73d St., New York.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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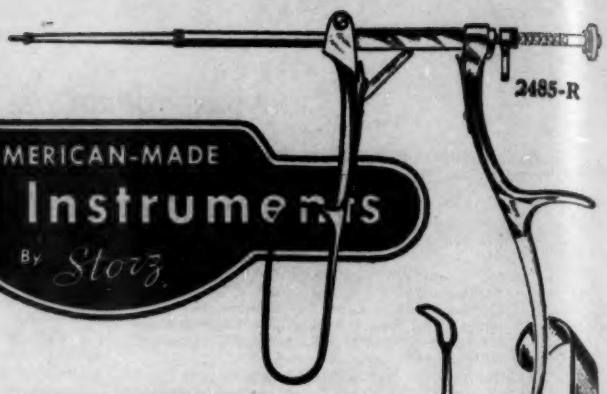

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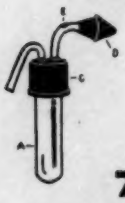

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